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EDITORIAL NOTICE.

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ARCHIVES OF OPHTHALMOLOGY.

THE BASIC PRINCIPLES OF DIAGNOSIS IN MOTOR ANOMALIES OF THE EYE.[1]

BY DR. ALEXANDER DUANE, NEW YORK.

OUR tests for anomalies of the ocular muscles must obviously be based on an answer to the following questions:

1. What are the normal movements of the eyes singly and in conjunction?

2. What are the possible perversions of these movements, and what distinctive evidence of its presence does each particular perversion afford?

3. What are the most ready means of bringing these distinctive evidences to light?

We may subdivide the first question into the following queries:

(*a*) What are the actions of the individual muscles of the eye?

(*b*) How are these coördinated to move each eye by itself?

(*c*) What are the coördinated movements of the two eyes acting together?

ACTIONS OF THE INDIVIDUAL MUSCLES.

The actions of the individual muscles are ascertained partly from a consideration of their anatomy, but mainly through clinical observation, particularly in cases of paralysis. Most of the facts are well known; some perhaps are yet a little in doubt.

[1] Read by invitation before College of Physicians, Philadelphia, October, 1918.

According to my information and observation they are these:

The *external rectus* moves the eye straight out, the *internal rectus* straight in. In the nature of things the action in either case is less powerful toward the end of the movement and would seem to require reinforcement.

The *superior rectus* elevates the eye, adducts it, and intorts its vertical meridian. The elevating action in the primary position is considerable, and increases very decidedly as the eye is abducted. Theoretically it should be at its maximum when the eye is abducted 20° or so, but as a matter of clinical observation, it increases right up to the limit of movement, being at its maximum when the eye is turned far out. Similarly, it diminishes as the eye is adducted and becomes practically nil when the eye is turned well in.

The adducting and torsional actions of the superior rectus seem to vary considerably in different cases. Its torsional action offsets that of the inferior oblique, and in the primary position must be equal to it, for when the eye moves straight up its vertical meridian undergoes no tilting. Both adducting and torsional actions diminish to zero as the eye is abducted, and increase, though not greatly, as the eye is adducted.

Precisely the same is true of the *inferior rectus*. This muscle depresses the eye considerably in the primary position, and the depressing action increases continuously and fast as the eye is abducted, so that it is at its maximum when the eye is turned far out. On the other hand, it diminishes rapidly as the eye is adducted. The inferior rectus also adducts the eye and tilts its vertical meridian outward. The adducting and torsional actions, which seem to vary a good deal in different cases, diminish as the eye is turned out and increase moderately as it is turned in. The torsional action, which sometimes at least is marked, offsets that of the superior oblique and is at least equal to it in the primary position.

The *superior oblique's* main action is that of a depressor, its intorting action, upon which so much stress has been laid, its abducting action being quite subsidiary and often but little marked. In the primary position its action as a depressor is moderate—we may perhaps put it roughly as one third that of the inferior rectus—and it diminishes fast as the eye is ab-

ducted. Per contra, it increases rapidly as the eye is turned in. The contrary is true of the torsional and abducting actions, which increase, but not very fast, as the eye is turned out.

The main action of the *inferior oblique*, as we have learned from observations based on a series of complete tenotomies of the muscle, is to elevate the eye. In the primary position the amount of elevation produced by this muscle may vary from 3° to 15° of arc. At least, a deflection of this amount—or a reduction of a hyperphoria by from 5 to 25 centrads—is produced by the tenotomy. The elevating action increases fast as the eye is adducted and decreases fast when the eye is abducted. The inferior oblique also extorts and abducts the eye, particularly when the latter is already abducted. The abducting power seems not to be very great even at its maximum—at least, no marked lateral diplopia is caused by tenotomy of the muscle. The torsional effect varies. As a rule it seems to be slight or may be practically nil. I recollect only one case in which a marked torsional effect was produced by the tenotomy.

MONOCULAR MOVEMENTS.

From the foregoing statements it is clear that each eye has three abductors, the external rectus and the two obliques; three adductors, the internal, superior, and inferior recti; two elevators, the superior rectus and inferior oblique; two depressors, the inferior rectus and superior oblique; two muscles rotating the vertical meridian inward, the superior rectus and superior oblique; and two rotating the vertical meridian outward, the inferior rectus and inferior oblique. This synopsis, however, does not give a clear idea of the way in which these muscles actually coöperate in moving the eye. This is better shown by a fuller analysis.

The possible directions in which the eye can move are: straight out, straight in, straight up, straight down, up and out, up and in, down and out, and down and in. All clinical testimony shows that these movements are effected in the following way:

Movement Straight Out.—When the eye begins to move out from the primary position, the external rectus receives a strong stimulus and, according to Sherrington's law, the internal

rectus is inhibited and relaxes. At the same time that the external rectus receives its extra stimulus, all the other muscles except the internal rectus are stimulated too. The superior and inferior recti pulling, the one up and the other down, balance each other in vertical action, so that the eye does not swerve from its horizontal path but is kept steadily in it by the counteracting tension of the two muscles. Both muscles act to oppose each other in torsional action, so that the vertical meridian is held vertical. Both muscles combine to adduct the eye, so as to oppose to a moderate degree the outward movement, and doubtless by their steady pull prevent its taking place too suddenly or jerkily. The two obliques also counteract each other, so far as torsional and vertical actions are concerned, and thus also act to steady the eye in its course and prevent it from swerving up or down or from tilting to right or left. Both abduct the eye and so reinforce the action of the external rectus. In this stage of the movement their abducting action is in general about neutralized by the adducting action of the superior and inferior recti. The net result is that at the beginning of the outward movement the eye is abducted strongly by the external rectus, which is then acting at a considerable mechanical advantage, and it is simply steadied in its course by the action of the other four muscles; the superior and inferior recti, in particular, having the important effect of checks or brakes to prevent the abduction from going on too suddenly.

As the movement progresses the external rectus acts at a greater mechanical disadvantage, and, moreover, is now opposed by the increasing passive pull of the lengthening and tightening internal rectus. If these two muscles alone were acting, the outward movement would be slowed up and impeded. But, as an offset to the diminishing power of the external rectus, the abducting action of the obliques now comes more and more into play, since their action in this regard increases as the eye is abducted, and, what is more important, the counteracting adducting action of the superior and inferior recti falls off altogether. The result is that when the eye has been turned out some 30° it is carried further out not only by the external rectus, which now acts somewhat less effectively than before and begins to feel the opposing pull of the stretched

internal rectus, but also by the obliques, whose abducting action is constantly increasing and is no longer opposed by the counteracting pull of the internal and superior recti. The eye is now kept straight in its path solely by the superior and inferior recti, which act simply to elevate and depress the eye and whose opposing vertical actions keep pace with each other as they steadily increase. On the other hand, the vertical meridian is kept vertical solely by the opposing, equal torsional action of the two obliques.

Movement Straight In.—Quite a similar coöperation of muscles obtains in inward movement. At the outset of the movement the external rectus is inhibited and relaxed and all the other muscles receive an extra stimulus. The internal rectus, acting at a considerable mechanical advantage, moves the eye strongly inward, excessive and jerky movement being prevented by the counteracting pull of the subsidiary abductors, *i.e.*, the superior and inferior obliques. The inferior and superior recti, on the other hand, by their adducting action, which about balances the abducting action of the obliques, reinforce the inward movement. The eye is kept from swerving up or down and the vertical meridian is kept vertical by the combined and opposing actions of the superior and inferior recti and the obliques.

When the eye is turned well in, the internal rectus, acting now at some mechanical disadvantage, is opposed mainly by the passive pull of the stretching external rectus, since the abducting power of the obliques has now become nil; and it is sensibly reinforced by the steadily increasing adducting action of the superior and inferior recti. The eye is kept from swerving up or down solely by the obliques, and its vertical meridian is kept vertical solely by the superior and inferior recti.

Movement Straight Up.—When the eye is moved straight up, the inferior rectus and superior oblique are inhibited and relaxed, and the other four muscles are stimulated. The eye is carried up by the combined action of the superior rectus and inferior oblique, the former predominating. The lateral actions of these muscles oppose each other and tend to keep the eye steady in its upward course. The like is true of the external and internal recti, which contract strongly and by their equal, opposing actions keep the eye from swerving to

the right or left. The vertical meridian is kept vertical by the counteracting torsional actions of the superior rectus and inferior oblique.

Movement Straight Down.—Similarly, in a movement straight down the superior rectus and inferior oblique are relaxed, and the eye is carried down by the inferior rectus and superior oblique. The abducting and adducting actions of the latter muscles counteract each other, as do the abducting and adducting actions of the external and internal recti, so that the eye is kept from swerving to right or left as it goes down. Moreover, as the torsional actions of the inferior rectus and superior oblique nullify each other, the vertical meridian is kept vertical.

Movement Up and Out.—When the eye moves up and out, the internal rectus, inferior rectus, and superior oblique are relaxed. The eye is carried out by the external rectus, the movement being opposed at first by the superior rectus and reinforced by the inferior oblique. As the eye goes further out, and the opposing action of the superior rectus falls away, the reinforcing action of the inferior oblique increases, so that the outward movement is hindered only by the passive pull of the stretching internal rectus. At the beginning of the movement the eye is carried up by both the superior rectus and inferior oblique, but soon the latter ceases to act, and the superior rectus alone elevates the eye. The vertical meridian, at first really vertical, later becomes tilted outward, since the increasing torsional action of the inferior oblique is now unopposed by the superior rectus, which is acting solely as an elevator.

Movement Up and In.—When the eye moves up and in, the external rectus, inferior rectus, and superior oblique are relaxed. The eye is carried in by the internal rectus, the movement being opposed by the inferior oblique and reinforced by the superior rectus. As the eye goes further in, the reinforcing action of the superior rectus increases and the opposing action of the inferior oblique falls away, so that the inward movement is opposed only by the passive pull of the lengthening external rectus. The eye is carried up at first by the superior rectus and inferior oblique together, later by the oblique alone. The vertical meridian is tilted in, because the increasing torsional

action of the superior rectus predominates more and more over the decreasing torsional action of the oblique.

Movement Down and Out.—When the eye moves down and out, the internal rectus, superior rectus, and inferior oblique are relaxed. The eye is carried out by the external rectus, assisted more and more by the superior oblique and counteracted to a decreasing extent by the inferior rectus. It is moved down at first by both inferior rectus and superior oblique, later by the former muscle alone. Its vertical meridian is tilted in by the increasingly predominant torsional action of the superior oblique.

Movement Down and In.—When the eye moves down and in, the external rectus, superior rectus, and inferior oblique are relaxed. The eye is carried in by the internal rectus, assisted more and more by the inferior rectus and counteracted to a decreasing extent by the superior oblique. The eye is carried down at first by both inferior rectus and superior oblique, later by the superior oblique alone. The vertical meridian is tilted outward by the increasingly predominant torsional action of the inferior rectus.

We see therefore that movements straight out and straight in are effected by the coöperation of five muscles; movements straight up and straight down by the coöperation of four; and oblique movements by the coöperation of three. Furthermore, it is only in movements straight up and straight down that the muscles exert a constant form of action throughout the eye's excursion. In lateral and oblique excursions, the actions of all the muscles involved except the internal and external recti keep changing in character as the eye advances. It is owing to this fact that the two lateral and four oblique, but not the two vertical directions of the gaze, afford distinctive evidences of the nature of the trouble when any given muscle is paralyzed or overacting. Since these two lateral and four oblique directions of movement are the only ones of diagnostic importance, they are denominated the *cardinal directions of the gaze* (see infra).

BINOCULAR MOVEMENTS.

What, however, as clinicians we are really concerned with is not the muscles nor even the movements of either eye alone,

but the coördinated movements of both eyes together. It is important to remember this fact, as it constitutes the basis of both our tests and our treatment.

The binocular movements that we have to consider are:

(*a*) Parallel or conjugate movements.

(*b*) Convergence.

(*c*) Divergence.

The other possible coördinated movements of the eyes are clinically of small importance.

Conjugate Movements.—For purposes of diagnosis it is convenient to divide the conjugate movements into movements to the right, left, up and right, up and left, down and right, and down and left. Corresponding to these six cardinal directions of the gaze are six pairs of muscles, each pair having a distinct field of action in which it is predominant, so far at least as its main action (abduction, adduction, elevation, or depression) is concerned. Thus we have the following:

Field of Action	*Muscles Predominant in Field*	
Right	R. external rectus	L. internal rectus
Left	R. internal rectus	L. external rectus
Up and right	R. superior rectus	L. inferior oblique
Up and left	R. inferior oblique	L. superior rectus
Down and right	R. inferior rectus	L. superior oblique
Down and left	R. superior oblique	L. inferior rectus

We thus see that the twelve muscles of the two eyes are divided into six pairs of associates. Thus the superior rectus of the right eye is the associate of the inferior oblique of the left. Now it must be emphasized as the basis of all of our tests of coördinate movements that of the two muscles constituting a pair of associates either one will carry its eye as far and as fast in any given direction as the other member of the pair will carry the fellow eye, and that, further, each pair of associates has as its field of predominant action one of the six cardinal directions of the gaze, which belongs to it and to it alone.

Convergence.—Convergence is effected by an equal simultaneous contraction of both interni, due to an impulse that emanates from a center distinct from that which causes a conjugate movement of either eye inward. This distinction between convergence-adduction and conjugate-adduction is of great importance in the diagnosis of motor anomalies.

Divergence.—Divergence is effected by a simultaneous equal relaxation of both interni, associated in all probability with an active contraction of both externi.

VARIETIES OF MOTOR ANOMALIES.

A motor anomaly consists of some perversion of a coordinate movement. We have therefore as the fundamental varieties of motor anomalies:

(*a*) Affections of conjugate movements.

(*b*) Affections of convergence.

(*c*) Affections of divergence.

Affections of Conjugate Movements.—The affections of conjugate movements comprise:

1. Those located in the muscles themselves or in the nerves and nerve nuclei directly supplying them. These include:

Muscular insufficiency (in the proper sense of the term).

Paralysis and spasm of the individual muscles.

2. Those located in the centers presiding over conjugate movements and their connections. These include:

Conjugate paralysis and spasm.

Nystagmus.

The characteristic mark of a muscular insufficiency, paralysis, or spasm is that, since some individual muscle is under- or over-active, the affected eye will fall behind or shoot ahead of the other when the two enter the field of action of the muscle concerned, *i.e.*, when they attempt to look in a certain one of the six cardinal directions of the gaze. This will be evidenced by a visible lagging of one eye, a corresponding deviation shown by the screen and other tests, and by diplopia—all these symptoms increasing progressively as the eyes are carried in a given direction.

The characteristic mark of a conjugate paralysis or spasm or of nystagmus is the presence of a bilateral equal deflection of

the eyes from the primary position or a bilateral and equal restriction of movement.

Affections of Convergence.—Affections of convergence comprise:

Convergence-excess.

Convergence-insufficiency.

Their characteristic mark is a deviation which increases as the object looked at is approximated to the eyes, but which does not increase as it is carried to the right or left.

Affections of Divergence.—Affections of divergence comprise:

Divergence-excess.

Divergence-insufficiency.

Their characteristic mark is a deviation which increases as the object is carried away from the eyes and which does not increase as the object is carried to the right or left.[1]

Mixed Conditions.—We very often find mixed conditions, including:

Convergence anomalies with consecutive divergence anomalies.

Divergence anomalies with consecutive convergence anomalies.

Either the one or the other, singly or combined, associated with muscular insufficiency, paralysis, or spasm.

These various pathological conditions, simple and mixed, we usually group under the two heads of heterophoria and squint. These two are often regarded as quite distinct in nature, but according to my belief and experience there is no essential difference between them. It is true that the symptoms, prognosis, and treatment differ somewhat, but this, I am sure, is due simply to the fact that ordinary squint represents a later and more complex stage of the process, of which heterophoria is the first and simplest expression—the two conditions themselves being in fundamentals the same.

Types of Heterophoria.—The heterophorias comprise esophoria, exophoria, and hyperphoria.

[1] Provided that in carrying the object to the right or left the angle subtended by the two visual lines remains the same. This requires that the object should be carried to the right or left along the circumference of a horopteric circle, *e.g.*, round the circumference of a horizontal hoop, the near part of which is held in contact with the root of the patient's nose.

The *esophorias* that we ordinarily encounter are cases of convergence-excess, which may be complicated by a consecutive divergence-insufficiency. In a small minority of the cases they are due to a primitive divergence-insufficiency or to actual weakness of the externi.

About two thirds of the cases of *exophoria* are due to convergence-insufficiency, complicated quite often with a consecutive divergence-excess. In one third of the cases, divergence-excess is the primary condition. Quite often such a primary divergence-excess persists indefinitely without increase or complication. In other cases it is followed sooner or later by a convergence-insufficiency. The distinction between the two types is important, as their treatment differs. Moreover, the more the element of convergence-insufficiency enters into the condition, the more marked usually are the symptoms.

The *hyperphorias* are probably in almost all cases due to insufficiency or actual paresis of the individual muscles. Exception may be made of certain cases of hyperphoria, sometimes considerable in amount, occurring as the result of wearing prisms or ill-fitting glasses which act like prisms. In this case the hyperphoria is due to an active spasmodic divergence of the eyes in a vertical plane.

Types of Squint.—Squint develops out of a heterophoria or a paresis. As already intimated, this statement is counter to the belief of many, who hold that squint and heterophoria are basically different and that the former is not the outcome of the latter. But all my experience indicates the correctness of the view enunciated.

According to this, the *usual type of the convergent squint* of childhood starts as an esophoria due to accommodative convergence-excess. This esophoria is gradually converted into a squint, at first intermittently periodic, then constantly periodic, and finally continuous. Later still, changes take place in the muscles themselves (actual weakening of the externi, contracture of the interni), aggravating the squint and rendering its cure more difficult.

So, too, the *usual type of a divergent squint* starts as an exophoria due either to a convergence-insufficiency, usually accommodative in origin, or, in a fair proportion of cases, to a divergence-excess. The deviation, as in the case of a con-

vergent squint, passes through the successive stages of a heterophoria, a periodic squint, a continuous comitant squint, and finally a squint no longer strictly comitant because it is complicated by consecutive muscle changes.

As against these, which constitute the great majority of cases of squint, must be set two other types. First, there are the ***paretic squints***, of which the congenital squints of childhood compose a specially important variety. Of these latter there are various forms, but two in particular deserve attention. One is the peculiar syndrome characterized by complete inaction of the externus, restricted action of the internus in the same eye, and retraction of the eyeball and narrowing of the palpebral fissure in adduction, with often an upshoot of the inturning eye. This is rather rare. Much more frequent and important is the congenital paresis of the superior rectus, associated in most cases with upshoot of the other eye in adduction, due to overaction of its inferior oblique.

The third variety of squints is the ***mixed type***, in which a congenital paresis, especially one of the sort just mentioned, is followed by a gradually developing convergent or divergent squint. The lateral deviation in this case is not only consecutive to the vertical deflection, but is evidently dependent upon it. These cases are fairly numerous and are important, since their treatment demands that the vertical deviation shall be made out and shall be remedied by operation before any effective treatment can be applied to the lateral squint. I have seen several cases in which neglect of this fact has produced disappointing results. On the other hand, if the vertical deflection is removed, to start with, the lateral squint may disappear of itself.

TESTS TO BE USED AND WHAT THEY DETERMINE.

The tests used in order to make the diagnosis may be very simple, if we have regard to the facts just enunciated concerning the physiology and the pathology of the eye movements. Our tests, in fact, have a twofold object: first, to determine whether any deviation present is due to an anomaly of conjugate movement, convergence, or divergence, or represents a mixed condition; and, second, to ascertain how far and

under what conditions this deviation impairs the ability of the two eyes to move in harmony and to see as one. In determining the first point we must in particular ascertain whether the deviation increases in any special direction of the gaze or increases in convergence or in divergence. My own procedure is as follows:

1. First, I determine by the screen test, parallax test, and phorometer the ***amount and kind of deviation for distance and near***. The screen test at the same time discloses whether the deviation is a heterophoria or a squint and, if the latter, whether it is alternating or monocular and whether it is comitant or intermittent.

If the deviation is notably greater for distance than for near, we should infer that it is mainly due to a divergence anomaly (to a divergence-excess if it is an exophoria, divergence-insufficiency if it is an esophoria). This inference will be confirmed if it turns out that the deviation does not increase in looking to the right or left.

If the deviation is notably greater for near than for distance, we are in general dealing with a convergence anomaly (convergence-excess, if it is an esophoria, convergence-insufficiency,[1] if it is an exophoria).

If the deviation is the same for distance and near we are dealing with a mixed condition.

2. I determine the ***prism-divergence*** for distance, *i.e.*, the ability to overcome prisms, base in, when the patient is looking at an object across the room. If this is high (nine centrads or over) we would infer a divergence-excess (primitive or consecutive); if it is low (below three centrads or actually negative[2]) we should infer a divergence-insufficiency. The diagnosis in either case would be confirmed if the deviation did not increase in looking to the right or left.

3. I determine the ***convergence near-point***, measuring it in the way I have elsewhere described[3] from the base-line con-

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[1] Some cases of moderate weakness of the interni when associated with good converging power may give a marked exophoria for near. These cases are marked by the fact that the convergence near-point is not remote as it is in a true convergence-insufficiency (see infra).

[2] That is, if there is homonymous diplopia.

[3] Arch. of Ophth., xliii., No. 5, p. 476.

necting the centers of rotation of the two eyes. If the near-point is remote (over 100*mm* from the base-line) we argue the presence of a convergence-insufficiency.

It will be noted in some cases of divergent squint that the patient, while never actually converging so that both eyes fix the approximating object, yet makes a strong effort to do so, the eyes turning in more and more as the object is brought closer, until some comparatively near-point is reached, when the effort is given up. The presence of such a ***relative near-point of convergence*** should be noted, as when close it indicates good converging power and makes the outlook for operative or other treatment more favorable.

The presence of a low degree of exophoria for near with a remote convergence near-point indicates a marked convergence-insufficiency, while a high degree of exophoria for near with a comparatively close convergence near-point rather indicates a moderate weakness of the internal recti associated with high converging power.

4. I note the ***excursions of the two eyes*** when they are following a light-colored pin carried in each of the six cardinal directions of the gaze, observing whether one lags behind the other or shoots ahead of it when the latter is fixing. This test is advantageously made first with both eyes following the object; afterward, with one eye following and the other screened in such a way that the observer can see its action behind the screen.

This test I often supplement by determining the ***screen-deviation in each of the six cardinal positions***, a dot on a card being used in this case as the object of fixation; and, if diplopia is present, by mapping this on the ***tangent-curtain*** in each of the six positions.

These excursion and diplopia tests enable us at once to say if there is any affection of the conjugate movements (*i.e.*, any insufficiency, paresis, or spasm of the individual muscles), and to ascertain the muscle involved. For, if one eye follows the other without sensible lagging in each of the six directions and if the deviation, vertical or lateral, does not increase notably in any one of the six, then the deviation is comitant and must be due almost certainly to a convergence or divergence anomaly. Exception may be made of certain cases of old paretic

squint in which through contractures of the muscles or other complicating factors the picture of a comitant deviation is simulated.

On the contrary, if in any one of the six cardinal directions of the gaze one eye lags behind the other, or one eye when screened shoots ahead of its fellow which is fixing, or the screen deviation increases, or diplopia develops and grows progressively greater, we are dealing with a paresis or spasm of one of the two muscles constituting the pair of associates, whose field of action lies in that particular direction of the gaze.

The diagnosis of the special muscle affected can be made almost at a glance. The following table is as convenient as any for the purpose:

Lagging, Deviation, or Diplopia Increasing	*Character of Diplopia or Deviation*	*Muscle Affected*
To right	Homonymous	R. external rectus
	Crossed	L. internal rectus
To left	Homonymous	L. external rectus
	Crossed	R. internal rectus
Up and right	L. hyperphoria	R. superior rectus
	R. hyperphoria	L. inferior oblique
Up and left	R. hyperphoria	L. superior rectus
	L. hyperphoria	R. inferior oblique
Down and right	R. hyperphoria	R. inferior rectus
	L. hyperphoria	L. superior oblique
Down and left	L. hyperphoria	L. inferior rectus
	R. hyperphoria	R. superior oblique

In using this or any similar table there are several facts to be borne in mind.

(*a*) To make from it a sure diagnosis of insufficiency or paresis, the deviation, lagging, or diplopia must increase decidedly and progressively as the eyes are carried in the given direction. The more rapid the increase, the more marked the paresis.

(*b*) Theoretically, paresis of the superior or inferior rectus should be associated with crossed diplopia or deviation, and

paresis of an oblique with homonymous deviation. But the adducting power of the superior and inferior recti and the abducting power of the obliques are so feeble that the lateral diplopia due to paresis of these muscles is often neutralized or reversed by a diplopia due to other causes. Even in this case, however, the paresis causes the diplopia to change in a characteristic way. Take, for example, a paresis of the right superior oblique associated with a marked convergence-insufficiency. The crossed diplopia due to the latter may completely neutralize the homonymous diplopia due to the paresis of the oblique. But in this case the crossed diplopia, instead of remaining the same when the eyes are swung from left to right, as it would do if caused by an uncomplicated convergence-insufficiency, diminishes as the eyes are carried to the right and increases as they are carried to the left, because in the former position it is more and in the latter position it is less neutralized by the changing homonymous diplopia due to the paresis of the oblique.

An analysis of all possible cases gives the following interesting laws, which I enunciated a number of years ago.

An homonymous diplopia (or deviation) increasing to the right[1] indicates a paresis of one (and it may be any one) of the six muscles of the right eye[2]; or it may indicate a spasm of any one of the six muscles of the left eye. If the increase is rapid it indicates a paresis of the right external rectus.

A crossed diplopia (or deviation) increasing to the right indicates a paresis of some one of the six muscles of the left eye; or it may indicate a spasm of some one of the six muscles

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[1] Obviously the converse of this is true; *i.e.*, in this and the succeeding propositions "left" may be substituted for "right," and vice versa.

[2] For an homonymous diplopia increasing to the right may be due to paresis of an abductor whose abducting action increases to the right (right external rectus, right superior oblique, right inferior oblique) or to a condition causing a preponderating comitant homonymous diplopia combined with paresis of an adductor whose adducting action diminishes to the right (right internal rectus, right superior rectus, right inferior rectus). But since the external rectus is the only muscle that produces marked abduction and since to the right of the median line the three adductors of the right eye act very feebly, a *marked* increase in the homonymous diplopia in looking to the right must be due to paresis of the external rectus.

of the right eye. If the increase is rapid, it indicates a paresis of the left internal rectus.

(*c*) An homonymous diplopia due to a convergence or divergence anomaly or to a paresis of a lateral rotator naturally tends to increase as the eyes are directed down, and diminish as the eyes are directed up, while the reverse is true of a crossed diplopia. Hence a crossed diplopia that increases in the upper field or even one that is confined to the upper field or to a corner of it is not per se evidence of paresis of an elevator; nor is an homonymous diplopia that is similarly confined to the lower field evidence of paresis of a depressor.

5. In squint cases I determine with the amblyoscope, bar reading, or the red glass, whether the patient has ***binocular vision***, and how far he can get and maintain ***fusion***. In doing this I try to determine to what extent he suppresses one image. Suppression may be partial, as when a patient fails to recognize the diplopia produced by prisms or a red glass, but can recognize the diplopia produced by the amblyoscope. Or he may recognize the diplopia produced by prisms only when the latter are of such a strength as to approximate or separate the images by a certain amount.

Such are the main tests I use. In some particularly obscure cases we may have to apply ***other tests*** with special refinements, but these are very rarely necessary. The reverse is much more often true; ***i.e.***, we can make a diagnosis ***without employing even all the tests*** above given. In very young children with comitant or paretic squint, we can usually determine the kind and amount of deviation and the muscles affected by making the simple excursion test in the six cardinal directions of the gaze and by making the screen test for distance and near in the primary position—getting the child to fix for a moment on a tiny bright light, a well-defined toy, or any other object that may serve to hold its attention. The diagnosis can be made in this way in a child of eight months. In older patients we should in addition to these tests determine the convergence near-point, the degree of binocular vision with the amblyoscope, and, if double vision is present, the field of diplopia on the tangent-curtain.

I cannot emphasize too strongly the importance, particularly in squint cases, of the screen and excursion tests. These

alone, properly made, will show in a few minutes whether we are dealing with a heterophoria or a squint, whether the deviation is due mainly to a divergence or convergence anomaly, or to a mixture of both, whether it is alternating or monocular, constant or intermittent, periodic or continuous; and finally whether it is strictly comitant, whether it is comitant but complicated by an insufficiency or paresis of an individual muscle, or whether it is strictly paretic. If the deviation is paretic, these same tests disclose the muscle or muscles affected.

Whether we follow precisely the methods above given or not is doubtless immaterial. But we must, at all events, pursue some such systematic scheme as this, based on the physiological factors involved, or we shall fail to interpret the case aright and shall not be in position to undertake a rational plan of treatment.

ILLUSTRATING DR. COHEN'S ARTICLE ON "PRIMARY INTRADURAL TUMOR OF THE ORBITAL PORTION OF THE OPTIC NERVE."

FIG. 1. Low power appearance of ganglion cells and hyperplasia of neuroglia tissue.

FIG. 2. Same as Fig. 1, under higher power.

PRIMARY INTRADURAL TUMOR OF THE ORBITAL PORTION OF THE OPTIC NERVE.[1]

By Dr. M. COHEN, New York.

(With four illustrations on Text-Plates I. and II.)

Miss R. C., age 18, parents died of tuberculosis. They left two children, the patient being one, the other a brother who is at present in the U. S. Army. The previous history is negative, except for complaints of frequent and severe headaches for several years. The patient is moderately under-sized, well nourished, intelligent, but apathetic. Her main symptom is increasing protrusion of the eye and marked disturbance of the vision, of the left eye. The protrusion she states has existed as long as she can remember. The vision also has been poor for a long time, and has been absolutely lost for the past six months.

Dr. Troncoso first saw the patient a month and a half ago, when he made the diagnosis of optic nerve tumor. The patient was admitted to the Post-Graduate Hospital on September 1st. A complete physical examination—including a neurological report, differential blood count, blood Wassermann, rhinological examination, and X-ray examination of the skull—was normal. General tuberculin reaction test was not considered advisable. The eye report before operation was as follows:

Right eye: external examination normal; vision equal to $\frac{20}{20}$; fundus normal. Left eye: vision nil, and without any direct pupillary reaction. The pupil was slightly dilated, circular and central. The eyeball was markedly exophthalmic, but without any inflammatory symptoms. The palpebral fissure was slightly widened. The eyeball was displaced straight forward, with no loss of ocular motility in any direction. Palpation of eyeball met with an elastic

[1] Presented before Section on Ophthalmology, New York Academy of Medicine, Oct. 21, 1918.

resistance. The fundus examination showed solely a post-neuritic optic atrophy; the retinal vessels were normal.

From these clinical facts the diagnosis of tumor of the optic nerve was made.

The Kroenlein operation was advised, only to conserve the eye and reduce the exophthalmos. It was consented to by the patient and relatives. Enucleation was considered but not suggested, as the size and nature of the tumor were unknown. The usual operation was performed. After access to the apex of the orbit, a mass was palpated with the small finger. This mass was large, smooth, globular, and boggy, and extended from about *6mm* from the eyeball to about *3mm* from the optic foramen. After the surrounding tissues were retracted, the tumor could be seen, readily. Deep catgut sutures were then passed around the apparently normal optic nerve, at both extremities, and then, with curved scissors, both extremities were cut on the tumor side. At the optic foramen end, the tumor was cut into slightly. The suture was then gently drawn out, and the optic nerve of normal caliber was cut. Both specimens were then fixed in formalin solution.

Unfortunately the small optic nerve section from the optic canal was misplaced. Five weeks after the operation, the general condition is the same; the headaches are still evident. The eye examination is as follows:

Right eye: vision equals $\frac{11}{16}$, corrected to $\frac{20}{16}$. External appearance, normal. The field of vision with the perimeter and using *10mm* white and colored squares shows a marked concentric contraction, the visual field measuring from 18 to 20 degrees around the fixation point. This condition probably existed before the operation, and is difficult to explain satisfactorily unless one considers it of congenital or hysterical origin. The left or operated eye shows the following condition: Complete ptosis; no exophthalmos. The position of eyeball is up and out and practically stationary, only slight lateral motility existing. There is a moderate chemosis of the lower fornix. The entire eyeball is anæsthetic. The pupil is irregular and the cornea is transparent. Considering these results at present, enucleation, from a cosmetic standpoint, would have been advisable. The fundus shows marked alterations which are of unusual interest. The disk is pinkish in color with its margin completely obliterated. The main retinal veins have a straight course, are dilated, and filled with light-colored blood, except in the periphery, where the vein becomes gradually empty. The retinal arteries are normal in character. Their central light reflex is prominent; at places they are interrupted, due to retinal exudates. The

ILLUSTRATING DR. COHEN'S ARTICLE ON "PRIMARY INTRADURAL TUMOR OF THE ORBITAL PORTION OF THE OPTIC NERVE."

FIG. 3. Gross appearance of tumor.

FIG. 4. Edge of the tumor showing pia, arachnoid, and dural sheaths.

choroidal vessels are distinctly evident, on account of the depigmentation in the choroid and retina.

A few of the choroidal vessels are clearly seen, and are of a white color, which is probably due to a lack of blood in the vessels, and not to any degenerative changes in the vessel walls.

Numerous small oval-shaped whitish exudates are scattered near the macular area; larger greenish colored exudates are seen over various areas of the fundus. A large white patch, sharply but irregularly outlined, with retinal vessels coursing over it, is seen on the nasal side of the disk. This latter area is probably due to a partial retinal atrophy. The retinal and choroidal atrophy is quite diffuse in some areas.

Scattered over the entire fundus, one sees heaps and patches of pigment, light and dark colored. This pigment is mainly present in the periphery, where it is darker. The fundus changes are progressing. No hemorrhages are present.

The numerous pathological changes in the fundus are primarily due to the cutting of the central artery and also some of the posterior ciliary vessels, thereby interfering with the internal and collateral circulation of the eyeball, which causes the inflammatory and the degenerative changes present in the retinal and choroidal tissue.

Pathological Report. Gross.—Specimen is a pale yellowish pinkish mass, the shape of an olive, weighing 4 grams and measuring 2.8 x 1.5*cm*, with a short thick stem about 6*mm* in length, which is optic nerve. The surface is smooth. The consistency is somewhat doughy. On section the tumor is seen to be covered by the dura which is fairly normal in appearance. The proximal end of the tumor appears to be a fairly normal optic nerve, rapidly widening into a loose arrangement, which is the tumor proper, showing a simple general longitudinal linear mixture of translucent gray streaks. One focal hemorrhage appears close under the meninges, and several smaller ones beneath the dura proper.

Microscopic.—No serial sections were made, but a few longitudinal sections were studied. Several photo-micrographs were made for illustration.

Dr. Ewing considers the tumor to be a ganglionic neuroglioma or glio-blastoma (Mallory), a tumor of epiblastic origin whose cells are differentiated into neuroglia cells with ganglion cells in addition.

The hæmatoxylin-eosin stain shows numerous ganglion cells scattered over parts of the section and also grouped together. Other fields are entirely free from these cells; in such places the bundles of optic nerve fibers do not appear

disturbed. There is a definite increase of neuroglia tissue. In areas where the ganglion cells are found, the normal optic nerve structure is not evident. Ganglion cells lie in masses of fibers; it is impossible to state whether these are optic nerve fibers or fibers from the ganglion cells or neuroglia without special staining.

The ganglion cells vary in size and shape, some having a large pyramidal shape, others being smaller, and still others have an elongated and distorted outline. All are large enough to be designated giant cells.

The mesoblastic tissue or capsule surrounding the tumor is somewhat thickened showing no exudate. Walls of the blood vessels are not normal, showing the effects of pressure. There is a moderate amount of œdema in the tumor itself. There are numerous capillary hemorrhages which are rather recent. The structure of the tumor at the posterior extremity suggests the possibility of extension into the intracranial portion of the nerve. The ganglion cells and the hyperplasia of the neuroglia classifies the tumor.

Diagnosis.—Ganglionic neuro-glioma. I am indebted to Dr. Meeker for the pathological report.

From the clinical and pathological study of the case, the growth is probably benign and primary in the optic nerve. It is reported for the reason that it is, no doubt, the first case observed that shows definitely the presence of numerous ganglion cells occurring primarily in an intradural tumor of the optic nerve. As yet the nature of these cells cannot be definitely determined; they are probably of embryonal origin. It seems likely that these cells demonstrate another link in the embryological connection between the structure of the retina and the brain.

EXPERIMENTAL STUDIES IN INTRAOCULAR PRESSURE AND TONOMETRY.[1]

By WILLIAM McLEAN, M.D., New York.

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(With three figures in the text.)

LITTLE did I realize, when I began my work on intraocular pressure that my experiments would direct me into the paths laid out for me to follow. I had been using the Schiotz and Gradle tonometers and believed their readings to indicate the intraocular pressure.

In using these instruments I discovered certain annoyances which it seemed to me could be abolished in a tonometer constructed along different lines.

1. The distance between the cornea of the eye under examination and the reading scale of the Schiotz or Gradle instruments renders it difficult for one person to see that the tonometer is placed correctly on the cornea, and make a reading of the scale simultaneously.

2. A translation of the obtained readings into millimeters on the chart according to the weight used is necessary and consumes time.

3. The delineation of the fine lines on the chart is very trying to the eyes.

4. There are times when the estimated pressure is so far divergent from the indication by the instrument that a delay occurs to change weights, after the reading has been started. (Some nervous patients become annoyed while in the recum-

[1] Paper read before Section on Ophthalmology, New York Academy of Medicine, April 15, 1918.

bent position by the delay caused in changing weights, when it has been ascertained that the estimated weight was inadequate for the pressure of the eye under examination.)

In this respect the Gradle instrument is an improvement on the Schiotz as with the Gradle the changing of weights is simplified.

5. I have observed in both these instruments that the plunger fits the footplate barrel so closely that fluid remaining on the cornea at the time of making a reading may be drawn up into the instrument by capillary attraction and the accuracy of the reading impaired.

(Paul Knapp of Switzerland observed this same possibility of error due to capillary attraction.)

In my tonometer the calibrations on the scale are in millimeters of Hg. as determined by actual manometer readings, using the testing apparatus described in this paper.

The scale is placed just above the finger piece and as close to the eye as possible so it can be more readily observed by the operator.

The plunger instead of fitting accurately has a space of one millimeter between it and the base, and is held in position by the rounded ends of three hardened steel bearings placed 120° from each other. This reduces friction to a minimum and also prevents capillary attraction of any fluid which might remain on the cornea.

All the readings on the scale from 10 to 130*mm* Hg. are made without change of weights.

Before the days of the Schiotz tonometer I had been estimating the intraocular pressure of patients by the prevailing method of finger palpation. Estimations of pressure if we translated them into *mm* of Hg. were based on the experiment of Wahlfors. His determination of normal intraocular pressure was 26*mm* Hg. which was in harmony with the experiments on the lower animals, made by many other experimentors. This is the only experiment on record in which an eye of normal tension has been connected directly to a manometer.

When the Schiotz tonometer was adopted a new conception of the normal intraocular pressure was introduced. The Gradle tonometer is a copy of the Schiotz and Dr. Gradle

wrote me that he used a Schiotz tonometer to determine the scale for his tonometer.

My experiments tend to revert to the standards as established by Wahlfors and the early experimentors rather than the newer scale put forth by the Schiotz tonometer.

Before constructing my tonometer I reviewed all the literature on the subject I could find.

The first reference I found was that of Mackenzie who in 1830 observed that a hardness of the eyeball occurred in glaucoma. The importance of the correct conception of the intraocular pressure has been admitted since the full force of Mackenzie's discovery became manifest, and we find in looking over the history of Ophthalmology, that investigators early appreciated the inaccuracy of the estimation of intraocular pressure by the digital method.

The manometer in actual connection with the interior of the eye and with a perfect technique is the only method known today of obtaining absolutely accurate conceptions of the intraocular pressure.

The first records of experiments in ophthalmo-manometry that I could find are those of C. Weber in 1850 but he fails to draw any conclusions as to the normal intraocular tension. Later Wahlfors made some experiments with the human eye in situ in connection with the manometer and in some correspondence I had with Prof. Fuchs he advised me that Wahlfors's conclusions were in harmony with mine.

Two types of instruments have been devised. One called the impression type, that which records the amount of indentation or dimpling by a known pressure, and the applanation type, that which records the amount of flattening of the eyeball to a known pressure.

As early as 1863 von Graefe constructed a tonometer which was of the impression type. At the same time and independently of von Graefe, Hamer under the direction of Donders constructed an impression tonometer which was designated to be placed directly on the eyeball. Readings of the von Graefe and Hamer tonometers were obtained by pressing the instrument with the hand against the eye under measurement. Dor recognized the unreliability of any method which employed the hand to produce the pressure, and proposed that

the tonometer should be suspended by a string and allowed to work on the eye by its own weight. Impression tonometers were also made by Donders, Monnik, Lazerat, Priestley Smith, Snellen, Hembold, and others. The Hembold instrument was constructed to determine the intraocular pressure of the two eyes of an individual at the same time.

The tonometers of Fick and Maklakoff are good examples of the applanation type. The Fick tonometer was constructed on the principle of a spring lever pressing against a flat plate of a definite area, applied over the sclera, and noting the tension necessary to produce a flattening of the sclera equal to the area of the plate. In this instrument the hand is the source of energy for the spring.

Ostwalt and Koster made some minor modifications of the Fick tonometer but retained the general principle of that instrument.

The Maklakoff tonometer differed from those already mentioned and was designed to be placed on the uncovered cornea, and rested on the eye by its own weight. The instrument consisted essentially of a weight which had on its contact surface a flat disk of polished glass 1*cm* in diameter. When the instrument is to be used a smear of coloring solution is applied to the free surface of the glass, and the instrument allowed to rest momentarily on the cornea. Contact with the cornea presses the coloring solution out, and thus a ring of solution is formed, the inside clear area of which is equal to the area of flattened cornea. A transfer is immediately made on prepared paper from which the intraocular pressure is computed.

Of the many tonometers brought out up to the time of the Schiotz instrument, the Maklakoff was perhaps the best suited for general use.

The usual method of determining the intraocular pressure experimentally has been to introduce a canula into the interior of the eye, and attach a manometer by means of tubing to the canula. Both the water and mercury manometers have been employed, depending on the construction of the apparatus.

Among the experimentors are: Weber, Leber, Neisnamoff, Pfluger, von Hippel, Grunhagen, von Schulten, Stocker, Starling, Adamuk, Holtzke, Hill, and Flack.

The experiments performed by these men were on dogs,

rabbits, and cats, and their conclusions are that the normal intraocular tension of these animals varies from 18 to 35*mm* of mercury.

The experiments of Weber and Wahlfors were on the human eye.

In constructing an apparatus for my experimental work on eyes in situ, I tried to incorporate the good features of the apparatus used by previous investigators with improvements of my own design. I made a canula having a tapered steel needle, the opening in the needle being on the side. The taper conformed to an angle of two degrees and had two flat sides. I made two needles, the lumen of the one used in the anterior chamber was .8*mm* in diameter, while for use in the vitreous a needle of 2*mm* bore was used.

To overcome any possibility of altering the original tension of the eye by the volume of metal thrust into the eye, a needle valve was interposed in the lumen just far enough back from the opening to compensate for the volume of the metal in the eye. A half turn of the valve was sufficient to completely open the needle.

Investigators have repeatedly found that canulas of small bore will become clogged when used in connection with the vitreous chamber. I discovered while working on the living eye that my needle of .8*mm* bore could not be depended on when it was in connection with the vitreous chamber, but it worked very nicely in the aqueous chamber. With the 2*mm* needle I had no trouble.

One experimentor has published his results of work done along the line of intraocular pressure with the Schiotz tonometer where he used a canula of .8*mm* bore on eyes of the dead human being. During life the pulsations imparted to the fluid of the testing apparatus which comes in contact with the contents of the interior of the eye is an indication that the canula has not become clogged. With dead eyes there is no pulsation to aid the experimentor, so it is impossible to know that the apparatus is working properly. I made these experiments before the paper referred to above was published. After reading the article, to satisfy myself, as to the value of experiments with an .8*mm* needle in the vitreous, I repeated the experiments and found they verified my first conclusions.

In my work the canula was connected to a thirty-inch-long glass capillary tubing by short lengths of rubber pressure tubing. The total volume of fluid contained in the capillary tubing was about ten minims. The other end of the glass tubing was connected through a T piece to a manometer and a reservoir. The object of the capillary tubing was to enable me, by means of a small air bubble sucked half way up the tubing, to compare and equalize the manometer and the intraocular pressures.

A water manometer was used, but it was graduated to its equivalent in millimeters of mercury. The reservoir was sus-

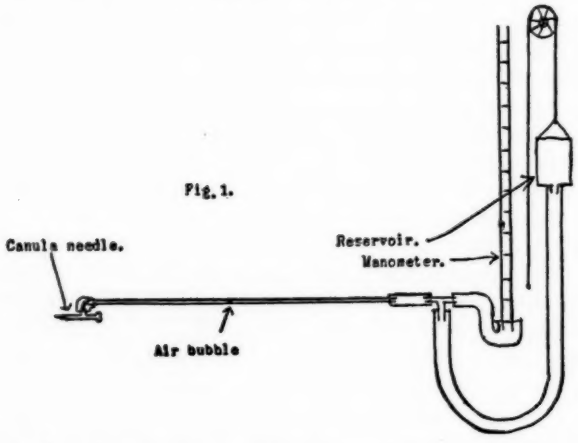


Fig. 1.

pended and was capable of adjustment as to height, in order to alter the manometer pressure at will. Fig. 1 shows a cut of the apparatus. Fig. 2 shows an enlargement of the canula needle.

In using the apparatus the needle was introduced with its valve closed, and the connected manometer registering the estimated intraocular pressure. The position of the air bubble was marked and when all was in readiness, the valve was given a half turn and the movement of the air bubble noted. If the bubble moved toward the manometer it indicated a greater intraocular than manometer pressure, and vice versa. A manometer pressure was obtained which caused

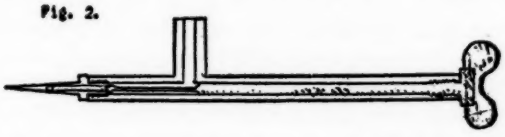
the bubble to remain at, or, in case of movement, to return to its original position. A reading of the manometer was then made.

Great care had to be exercised to prevent any traction or pressure of the needle on the eyeball.

To the present time, of living animals I have employed rabbits and dogs, with the exception of three living human eyes.

I have been particularly fortunate in being allowed to make experiments on three human eyes in situ. These eyes were all glaucomatous and were being removed on account of an uncontrollable pain. The experiments of two were performed

Fig. 2.



before I had my tonometer perfected. On the third both the Schiotz and McLean tonometers were used.

In my experiments, an observation was made, one which I have never read or heard of: that the tonometer gave a marked difference in intraocular pressure under local, and immediately after under deep general anæsthesia. The animal was first cocainized and the intraocular tension determined by tonometer, then after administering the anæsthetic the tonometer was again used before any other instrumentation was done. On the human cases observed the same proportion of difference prevailed.

I have not made enough observations of this difference in intraocular tension before and during deep general anæsthesia to do more than merely mention it as occurring among all the tests made.

I spoke to the late Dr. Marple of this observation and he noted it when he was present at one of the experiments. Later, in a conversation he had with another oculist, on the subject of the difference of ocular tension in local and deep general anæsthesia, he made the statement that he had since observed that difference in his operative work.

At the time most of these experiments were made I had not

finished perfecting my tonometer so the tonometer readings were made with Schiotz tonometers.

The tonometer readings on the human eyes were all made by Dr. Hallet. Dr. Marple was present when one of the tests on a rabbit's eyes was made and his Schiotz tonometer was used.

ANIMAL EXPERIMENTATION

First rabbit: The Schiotz tonometer registered 17.5 under cocaine and under ether 12. The small canula was introduced into the anterior chamber and a manometer reading of 33 was made. The anæsthesia was increased and the manometer pressure fell to 29.5, then 25, 23, 22, 22, 22. The anæsthesia was continued and made more profound from the time the canula was inserted until a continuance of the anæsthetic failed to further reduce the tension. The anæsthetic was then removed and the manometer pressure required to keep the air bubble at its mark had to be gradually increased and rose to 30*mm* when the animal showed signs of regaining consciousness, and the needle was removed.

The air bubble all the time showed pulsations which were synchronous with the animal's heart beat. Such pulsations are a positive evidence that there is free communication between the interior of the eye and the manometer.

Second rabbit: This animal was quite refractory before general anæsthesia was used so that we could not get a satisfactory reading under the local anæsthetic. When general anæsthesia was applied he succumbed rapidly and by the time we were ready he was quite deeply under the anæsthetic. The Schiotz tonometer then recorded 12*mm* Hg.

The canula was immediately inserted and the pressure per manometer was 19.5, then it dropped to 17, 16.5, 16.5, 16.5. No further drop in the tension occurred and the anæsthetic was removed. Shortly the tension began to rise and manometer readings were recorded at 21.5, 23.5, and later 25. When the manometer had reached 25*mm* the needle was removed on account of signs of returning consciousness of the animal.

Third rabbit: The Schiotz tonometer under cocaine and before ether anæsthesia registered 23. During ether 16.5.

The needle was inserted into the anterior chamber and a reading of 27*mm* was made. Then it dropped to 21, 19.5, 19.5, 19.5. The anæsthetic was removed and the tension rose as in the others, until it reached 26*mm* of manometer pressure.

Fourth rabbit: The Schiotz instrument under cocaine registered 21*mm* Hg. and as soon as the animal was judged sufficiently etherized a reading of 14 Schiotz was recorded. The manometer at that moment registered a pressure of 24*mm* and as the anæsthesia was deepened the intraocular pressure dropped, the lowest reading being 20.5*mm*. There seemed to be no further drop in pressure so the anæsthetic was removed and readings were recorded at 22, 27, 28, when the animal showed signs of recovery.

Probably this rabbit was deeper under the anæsthetic than we appreciated when we started our manometer readings, as the drop from the first to the low reading was less than the others except the second rabbit, which we knew was well under the anæsthetic when we started, and because on the recovery the intraocular pressure rose to 28*mm* by the manometer.

Fifth rabbit: The readings were O. D. 17 and O. S. 16 Schiotz, under cocaine, and when etherized readings of 8*mm* Schiotz were made on each eye. The canula of .8*mm* bore was introduced into the vitreous chamber of one eye. Soon after the needle was inserted the pulsations of the air bubble ceased, and the changes of manometer pressure were not attended with the usual quick response of the air bubble. The needle was removed and was found to be clogged with vitreous.

After cleaning the needle it was inserted into the anterior chamber of the other eye. The animal was then under deep anæsthesia and the manometer registered 15*mm* Hg. At this point the animal's breathing almost stopped and the anæsthetic was removed. Artificial respiration had to be resorted to and the animal partially revived, and a manometer reading was recorded at 16.5*mm*. The animal choked with mucus before other readings were made and all efforts to restore him failed.

Of the human eyes in situ used, the first case was one of total blindness in that eye from absolute glaucoma, and the

eye was being removed on account of uncontrollable pain. Tonometer readings were made on both eyes under holocain anæsthesia. The tension of the affected eye was 75 while the healthy eye registered 19 Schiotz.

Under ether the affected eye registered 45 and healthy eye 10 Schiotz.

A canula as described before with a bore of *2mm* was thrust into the vitreous chamber well back of the ciliary processes. After the position of the air bubble was marked, the valve was opened. It was found that a manometer pressure of about 65*mm* held the bubble at its mark. With the needle in the vitreous chamber we were able to make tonometer readings and compare them with the manometer, which readings were as follows:

Schiotz's tonometer	Manometer pressure
50	65
50	65
43	64
50	66

Tests made on this same eye after it had been enucleated gave the same ratio of variance between the manometer and Schiotz tonometer in all pressures of 10*mm* to 120*mm* Hg. as may be noted by Chart I.

On the second human eye tested, readings were made of the affected eye only. Before general anæsthesia a Schiotz tonometer reading of 88*mm* was made, and at the first reading under general anæsthesia the Schiotz recorded 72*mm* Hg.

The canula was immediately inserted and the Schiotz again recorded 72 which shows that the insertion of the needle made no change in pressure. The valve was opened connecting the interior of the eye with the manometer and a manometer pressure of 88*mm* was required to keep the air bubble in its original position.

As the anæsthesia was increased tonometer and manometer readings were made, which were:

Manometer	86	Tonometer	72
"	81	"	54
"	72	"	47

As the anæsthetic was continued there was no decrease of intraocular pressure below 72 (manometer) so the experiment was concluded and the eye enucleated.

CHART I

Human eye in situ used at the hospital for tests with water manometer and Schiotz tonometer. (Diag. Glaucoma Absolutum, O. D.) Manometer needle, *2mm* bore, with valve, and glass capillary tubing 30″ long and *1mm* bore. Needle was connected with vitreous chamber.

	O. D. Schiotz	O. S. Schiotz
Before anæsthesia	75	19
During "	45	10
	Manometer	
" "	50 65	
" "	50 65	
" "	43 64	
" "	50 66	

Same human eye (enucleated):

Manometer *mm*Hg.,	15 *Gm.*	10 *Gm.*	7.5 *Gm.*	5.5 *Gm.*
120	105			
110	96	86		
100	86	71		
90	58	65		
80	53	60		
70	42	56	62	
60	33	51	43	43
50	23	32	31	35
40	14	23	20	22.5
30	6	14.5	19	16
20	X	6	11.5	7.5

On the third human case both the Schiotz and McLean tonometers were used. On account of the conditions present we were allowed time only to make one reading each. In my haste I perhaps chose a faulty location for my canula as it was with difficulty that we could keep a connection of the interior of the eye open to the manometer as evidenced by the diminished oscillations of the air bubble, when as in the other cases the bubble oscillations were readily discerned all the time the valve was open.

The readings of this last case were, Schiotz 40, McLean 58, and the manometer recorded 60*mm.*[1]

[1] The demonstrations of the three human cases above cited were all made at the New York Ophthalmic Hospital in the presence of eye specialists. At the demonstration of the third case about thirty oculists were present.

It may be noted in all the experiments on the living eye, both human and animal, that the intraocular pressure was highest under local anæsthesia, and as soon as the patient was thoroughly under the influence of the ether the pressure was lowered; the deeper the anæsthesia the lower the pressure until a limit was reached.

In the animals we noted that the pressure rose, after the ether cone was removed, until it was back to its original pressure as the animal regained consciousness.

My aim in the work as shown in Chart II (experiments on dogs' eyes) was to find the relation of readings of the eyes in situ and immediately after enucleation. In this work the needle with the *2mm* bore seen in Fig. 2 was inserted into the vitreous chamber. The Schiotz readings before and after enucleation, and the corresponding manometer pressures are given side by side. It may be noted that the readings of the eye in situ agree very closely with those made after enucleation when connected with the apparatus I use for testing with enucleated eyes.

CHART II

Dogs' eyes. Tests made with Schiotz tonometer. Tests on eyes in situ, cocaine, then ether and tests on same eyes after enucleation.

Manometer	RIGHT EYE			LEFT EYE		
	In situ	Schiotz tonometer	Enucleated	In situ	Schiotz tonometer	Enucleated
96	77		81	80		
90	72		65	75		65
80	57		59	59		57
70	54		55	51		49
60	47		46	44		46
50	37		32	40		37
40	26		28	24		26
30	17.5		17	22.5		17
20	12		8	16		8
Cocaine	25			25		
Ether	13			22.5		

Chart I shows the readings made on the human eye in situ, and the corresponding readings after the eye was enucleated. It has been suggested that inasmuch as the large

number of tests I made on living eyes in situ agreed with the tests of those same eyes after enucleation it is pretty good evidence that my testing apparatus for enucleated eyes is fairly correct.

In my early experiments I tried the rubber dam apparatus suggested by Schiotz to be used in place of enucleated eyes, and found it entirely inadequate. I have learned since that Schiotz discarded this type of testing apparatus, and made the statement that he was sorry he ever brought it forth. To the present time I have found no satisfactory substitute for enucleated eyes in making the tests.

My testing apparatus for enucleated eyes consists of a water manometer, with its necessary connections of glass and rubber tubing; a canula connected to a stop-cock by short length metal tubing, to which is attached the apparatus which I have termed a pulsator.

I have used both the mercury and water manometers, and found the water manometer more convenient for use. The results were the same as of course they should be.

The canula I made with a *2mm* bore and made a trocar to fit, so that it could be inserted through a sufficiently long optic nerve into the vitreous chamber.

The connection between the eye and the stopcock is made entirely of metal to avoid the possibility of expansion by a more elastic substance.

The pulsator is an apparatus which I have constructed to simulate the pulsations of the arteries of the living eye. You have all noted in using the tonometer that there are oscillations of the needle which are synchronous with the heart beat. My apparatus causes these same slight oscillations of the indicating needle. The apparatus is run by clockwork.

THE TESTING OF MCLEAN TONOMETERS.

For testing tonometers, the enucleated eyes must not be kept in any hardening or preserving fluid and should be used as quickly as possible after enucleation. A few hours is sufficient to alter the resiliency of the tunics of the eyeball for testing purposes. They must be absolutely fresh.

While the eye is being used in the testing apparatus it is

ensheathed in a rubber cap which acts similar to Tenon's capsule. This cap is of extremely thin high-grade rubber tissue and is sufficiently large so as not to be put at all on the stretch when the eye is placed in it. It encases the eye almost to the margins of the cornea, and acts as a reservoir to hold the normal salt solution which keeps the eyeball from becoming less resilient.

Before using the eye it is cleared of all extraneous material down to the sclera. The imitation Tenon's capsule is adjusted

FIG. 3. McLean Tonometer.

and the canula with its trocar is passed in through the center of the optic nerve until the opening is in the vitreous chamber, when the trocar is withdrawn. A perfectly tight joint must be obtained, and the canula adjusted to the short metal tubing connected to the stopcock.

Outside the rubber tunic an adjustable ring support around the optic nerve acts as a base to support the eye without inducing any pressure or traction.

As soon as the eye is placed in position, I fill the rubber tunic with a normal salt solution. This keeps the eyeball

from becoming less pliable, as the eye under pressure changes quite rapidly by contact with the air.

When all is in readiness the petcock is opened. The pressure of the liquid should be raised in the manometer until the correct height is reached. (Do not proceed from a greater to a lesser pressure.) After the desired pressure is obtained an interval should elapse to allow the tunics of the eye to become adjusted to that pressure before a reading is made.

I have a solution of normal salt at hand, also a pipette with which I am constantly dropping the normal salt on the cornea before each application of the tonometer. The stopcock remains open, except when it is closed to make a tonometer reading. It is opened immediately after the reading and remains open until the next reading.

With this apparatus I am able to obtain any pressure from 0 to 135*mm* of mercury.

It was by actual tests with this manometer, using several tonometers on repeated readings, that the present scale on the McLean tonometer was adopted.

With this tonometer the lower limit of normal intraocular pressure is to be considered as 22, while I have not found a normal eye registering above 40. I have placed the upper limit at that figure because I have found eyes registering 40 in which there were no symptoms, subjectively or objectively, of glaucoma. It is difficult to give an absolute limit of normal tension. I have found a few eyes that registered 36 with the McLean tonometer and 22 with Schiotz in which symptoms of glaucoma were present.

Altogether in my experimental work I have tested nearly two hundred enucleated eyes, and a considerable number of eyes in situ of the lower animals.

Aside from the work of Weber and Wahlfors I think I am the only man who has done any experimental work on living human eyes, by connecting the interior of the eye to a manometer.

TWO RARELY DESCRIBED INSTANCES OF SCINTILLATING SCOTOMATA; SELF-OBSERVED.

By Dr. JAMES A. SPALDING, Portland, Maine.

DR. C. H. MAY in his *Text Book on Diseases of the Eyes*, eighth edition, page 292, and Casey Wood in his *Ophthalmic Therapeutics* at various places, speak of scintillating scotomata associated with symptoms of migraine, and probably due to circulatory disturbances in the occipital lobe. The scotoma, as they describe it, begins before each eye with a dark spot and spreads in scintillating and zigzag outlines (fortifications, embattlements) until there is a gap in the visual field, and occasionally the patient becomes temporarily blind. The phenomenon is always accompanied with headache or other symptoms of migraine, is probably due to eyestrain or physical exertion, occurs frequently, for a while, generally disappears in time, and is amenable to treatment, such as proper lenses, and rest of the eyes and body.

Although in practice for nearly fifty years, now, I have never seen a single instance of this sort in a patient, and for that reason it has occurred to me, that for the possible benefit of other oculists[1] it might be worth while to offer to the readers of the Archives a brief account of personal experience with two varieties of scintillating scotomata. And all the more so,

[1] It is to be hoped that the present taboo against the word "oculist" will ultimately cease, for it is burdensome to be compelled to resort to the euphemism of "ophthalmologist," "ophthalmic surgeon," or "ophthalmological practitioner" as has to be done by writers in order to steer shy of "oculist." Surely, there is no harm in plain "eye doctor" whilst the Latin word "oculist" is no more distasteful to the eye, or the ear, than words derived from the Greek.

since those which I have perceived have never been associated with headache, or migraine, or any physical symptoms.

When very young I was hit in the left eye with a piece of steel, so that the sight was damaged, and the right eye used almost exclusively for far and near ever since. Some years ago on awaking in the morning after considerable use of my eyes by electric light the night before with + 2 D. S., for presbyopia, the sight of the right eye was so much blurred that I could not tell the time by a clock which I kept near at hand, so as to know how much time I had to spare to get my breakfast and catch the morning boat from an island where I was summering. I had the eye tested and examined at once by two competent observers, who found that, without warning I had developed in this eye an astigmatism of + .25 cyl., axis 105°. This lens was prescribed and worn steadily. The media were declared to be normal and transparent. In a few weeks the astigmatism increased to + .75 and the axis swung up to 90°. Later on, the axis went over to 65° where it remained some months. Then the astigmatism decreased to + .25 D. C., and the axis after a few variations settled back to where it originally was marked, 105° where it has now remained some years without change. Meanwhile, hyperopia of + 1 D. has developed, and at present with + 1 D. S. and + .25 cyl., axis 105°, vision in O. D. is normal. During the winter months, however, when the skies are misty, and the sun runs low, the hyperopia increases to + 1.50 or even to + 1.75, the astigmatism remaining as of old. Presbyopia is + 3. As a result of these curious changes in the focus of this eye I was obliged for some years to keep on hand three sets of lenses, for any possible changes in refraction during a long game of billiards, or auction or prolonged use of the eyes. The left eye remains unchanged with vision about $\frac{1}{8}$ with + .75 D. S.

I have never tried to puzzle my consulting friends concerning these curious changes in refraction, but have fancied that as my health was perfect and media transparent, as repeatedly assured, these might be due to alterations in the position or relation of the crystalline lens to the retina from some irregular spasm of the ciliary muscle. The pupil, by the way, has always been circular, so far as I have been informed, or observed personally.

These details have been mentioned, as they may have some connection with the scotomata about to be described, as well as to offer suggestions for the management of patients who from the same reasons, or with the same symptoms as mine, feel dissatisfied with lenses carefully prescribed, and properly made but a short time before. In my own experience with patients, I must say that I do not recall such odd changes in astigmatic axes, as have been mentioned, but which, if observed by others, might account for those curious instances in which we find new patients complaining that they have consulted other oculists without obtaining satisfactory lenses. With such a pair of eyes, then, in which one is used and has been used unsparingly for years at innumerable literary tasks, let me now mention two varieties of scintillating scotomata observed during some years past.

Soon after beginning to wear the lenses first mentioned for constant use, I was at a wedding by the seashore, sitting on a piazza and looking out on the sparkling ocean shining brilliantly beneath a summer afternoon sun. Suddenly there appeared before me at the center of vision a black oval spot about the size of a bean. It changed gradually to a greenish center, and the margins began to be crenelated, and zigzagged, increasing by leaps and bounds until the entire field of vision was absorbed with a great glare of light. The center remained a pale Nile green, the fortification margins a brilliant sparkling blaze of sunlight. I was not blind, for I could see everybody about me, but I could not see to read, because I tried it at once using a book near at hand. This lasted about two minutes, and then the center of the scotoma began to clear and the outside waves of light to expand ever wider and wider apart and into the extremes of the visual field when they disappeared. In three minutes, as it seemed to me, I could see as well as ever. I had no other symptoms. This scotoma I have seen at intervals for years; it is generally oval with axis horizontal; it comes without warning but generally in warm weather and it never varies in size, color, or behavior. I never see it in the open country, but generally after looking at bright light from confined interiors of houses or in looking up, after using the eyes steadily in the daytime. I have never seen it by artificial light.

The second scotoma originates in an entirely different manner and differs, also, essentially from the former in various ways. It always follows physical labor in my garden such as weeding, raking the grass after cutting by the lawn mower, or hoeing down the path for weeds, the boy following with a rake. Most of this work is done, not by bending over, but by kneeling on a newspaper like a woman. No matter how hot it may be, nor how hard I work, there is no symptom from the sight until I finish and then go down the bulkhead stairs into the dimly lit cellar in order to avoid passing through the kitchen. Thereupon, the scotoma appears in an instant. It is, however, no longer oval as in the case of the other but kidney shaped, with a section cut out at the right, where as might be said the ureters fit in. At first very minute and black it expands rapidly, the center taking on a distinct rose color, with outside waves as before of glorious sunlight. Proceeding wave by wave on the outside of the kidney-shaped center, it progresses to the outermost parts of the field of vision and finally disappears, as it began, from the center. Whilst this is going on, it seems as if the circulation in the retina must be interrupted and that each outside wave of the scotoma represents an effort of arterial blood to push down into the retinal vessels, and to force, as it were, a path for blood and consequently for light and sight. Like the first scotoma, this second one has only a transitory effect on the vision, and the eyes can soon be used as before. Having observed, at last, that the second obscuration is due to contrasts of light, it is hardly necessary to add that in order to avoid its anxieties and discomforts I now avoid the contrast between the bright light of out of doors and the dim light of the cellar, and make my way indoors through the well-lit kitchen.

The kidney-shaped scotoma always lies more obliquely than the oval affair, and the depression is always to the right. The depression sometimes suggests the place seen in an apple for insertion of the stem. I cannot help emphasizing the difference between the Nile-green color of the center of the oval scotoma in comparison with the rose-colored center of the kidney shaped.

It seems to me that patients observing such appearances as these which I have here described would be extremely agitated.

and for the very reason that mine are invariably followed by perfect sight again, I have mentioned them in detail, so that other oculists if consulted for such could honestly quote such a paper as this, from a competent observer, and assure their patients that the symptoms would terminate favorably, although they might recur from time to time after too much bright daylight or overwork out of doors, and coming into shaded or darkened rooms.

Much as we all dislike to parade our personal symptoms, I have felt it for the best to offer brief notice of my own oddities in eye practice because they illustrate sudden astigmatism and curious changes of axis; hyperopia with alternations according to bright or clouded skies; two different scotomata: one with fortification margins and greenish center, the other oval with rose colored center, lasting a few minutes and leaving the vision totally unimpaired.

ILLUSTRATING DR. DUNN'S ARTICLE "A NOTE ON THE SURGERY OF THE MEIBOMIAN CYSTS."

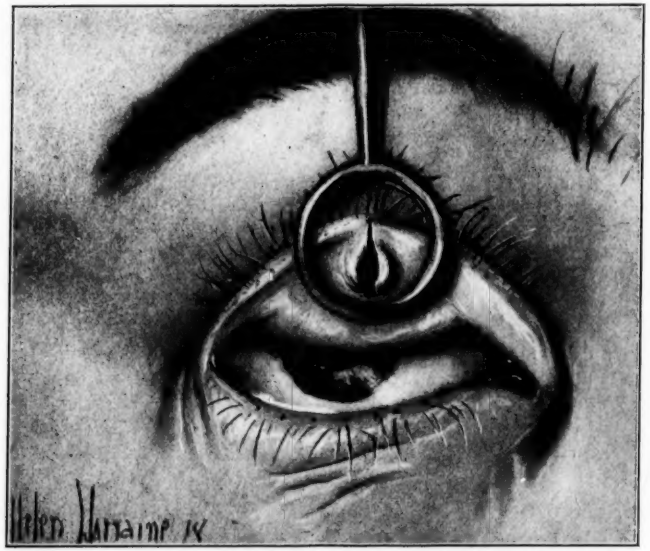


FIG. 1.—Completed operation, done after manner described.

FIG. 2.—Curette found to be most suitable.

A NOTE ON THE SURGERY OF THE MEIBOMIAN CYSTS.

By Dr. JOHN DUNN, Richmond, Virginia.

(With two illustrations on Text-Plate III.)

WEEKS, *Diseases of the Eyes*, p. 171, says: "The cyst should be opened, the incision being made on the conjunctival surface in the long axis of the Meibomian gland. The contents of the cyst should be curetted out. An antiseptic ointment may be introduced into the eye two or three times daily. . . . A cure may be effected in the course of a few weeks or months." So far as I know, the above is a good description of the generally practiced operation for the removal of a Meibomian cyst. The line of incision is well illustrated in de Schweinitz's *Diseases of the Eye*.

For many years this was the method I pursued, but always with the feeling that other oculists must be getting more satisfactory results than I did. Following this method there was always suffered by the patient what seemed to me an undue amount of discomfort; the curetted sac took always a longer time to heal than I thought ought to be the case in a part of the body so well supplied with blood vessels as are the lids; and, lastly, I felt no assurance that further treatment would not later be required. I knew these things were so, but I could not understand why the surgery of so common an affection, and one apparently so simple, had not been long ago perfected. The explanation came to me quite accidentally and with it what I now believe to be a solution of all the difficulties. My willingness to present this short paper came from having lately had a patient with an uncured Meibomian cyst which had been operated upon seven times. It was

cured at once by having recourse to the procedure to be mentioned.

The cyst, whether infected or not, should be split in the long axis of the gland, and the incision should always be carried *along the gland to and through its opening on the free border of the lid.* This is the important thing in the whole operation. Unless the affected gland be opened wide its whole length the operation will have been imperfectly done, for the proximal end of the gland, although apparently healthy, if not opened and curetted remains a possible source of infection and brings about all the troubles we have mentioned as occurring during the healing. This infection may already be present or may be acquired from the conjunctival sac during the course of healing.

My own method of doing the operation is as follows: Four drops of a one per cent. solution of novocain are injected beneath the skin of the lid overlying the Meibomian cyst. Three drops of a ten per cent. solution of cocaine are dropped against the conjunctival surface over the cyst; this is done twice at an interval of three minutes. At the end of ten minutes from the time that the novocain was injected an appropriate lid clamp is applied. A cataract knife is now inserted into the distal end of the cystic swelling, carried through and forced out of the opening of the gland on the free margin of the lid. The walls of the cyst, including the full length of the gland, are now curetted until they are smooth, after which they are wiped off with a solution of silver nitrate, fifteen grains to the ounce. The clamp is now removed and the patient is instructed to hold a piece of absorbent cotton firmly against the eye until the bleeding stops, after which a small amount of boric acid salve, five grains to two drams of white vaseline, is inserted into the conjunctival sac. The patient is told to put some of this salve into the eye three or four times a day. The results are all that can be desired.

ASTIGMATISM AGAINST THE RULE—ITS RECOGNITION AND FREQUENCY.[1]

By Dr. JOHN GREEN, Jr., and Dr. WILLIAM F. HARDY, St. Louis.

IT is universally recognized that the majority of astigmatic eyes have the meridian of greatest curvature at 90 degrees or within 45 degrees to the right or left of 90. Astigmatism with the meridian of greatest curvature in any position on the arc between these limits is called "astigmatism with the rule."

Conversely, astigmatism in which the meridian of greatest curvature is at 180 degrees or within 45 degrees, up or down, from 180 is designated inverse astigmatism or "astigmatism against the rule." Some writers limit the terms "astigmatism with the rule" and "astigmatism against the rule" to astigmatism in which the meridian of greatest curvature is, in the former case, at 90 degrees or within a few degrees of 90, and, in the latter, at 180 degrees or within a few degrees of 180. When the meridian of greatest curvature deviates decidedly from the vertical or horizontal, the astigmatism is called "oblique."

It is not always possible to determine just what are the boundary lines separating the three groups; and we suspect that different writers have used these designations in different senses. This is unfortunate, as it prevents a just comparison of statistics emanating from different sources.

DEFINITION OF ASTIGMATISM "WITH THE RULE" AND "AGAINST THE RULE."

In this study we have regarded all cases of astigmatism with the meridian of greatest curvature at any point on the

[1] Read before the Section on Ophthalmology, American Medical Association at Chicago, June 13, 1918.

arc from 46 to 134 degrees as astigmatism with the rule. All cases with the greatest curvature at any point from 0 to 44 degrees and from 136 to 180 degrees have been classified as examples of astigmatism against the rule.

Astigmatism with greatest curvature exactly at 45 or 135 degrees has frequently been classified as astigmatism with the rule. This classification seems to us purely arbitrary, as these cases might with equal reason be classified with astigmatism against the rule. Hence, we have omitted from consideration and classification the few cases of astigmatism we have encountered in which the principal meridians were at 45 and 135 degrees.

Our interest in the subject of astigmatism against the rule was stimulated in July, 1916, shortly after we began the systematic use of the astigmatic charts devised by Dr. Walter B. Lancaster.[1] Prior to this time, we had depended for the recognition and measurement of astigmatism on skiascopy, the test letters, the keratometer (Chambers-Inskeep), and various astigmatic charts. Of the latter we regarded as most satisfactory the wedge-shaped radii narrowing to the center, with the companion double-wedge crossing at right angles—Noyes's selection of Green's charts—and Verhoeff's astigmatic charts, which have concentric circles of fine lines and wider radii at 15-degree intervals, with the companion chart of wide lines at right angles crossed by finer lines. We have always made it a practice to search for the presence of astigmatism of the slightest amount at whatever axis, and have, of course, detected and corrected many examples of astigmatism against the rule. But it was not until we began the use of Dr. Lancaster's charts that we became impressed with the inadequacy of our former methods to detect and measure low grades of astigmatism. And it was not long before one interesting fact emerged, namely, that the number of cases of astigmatism against the rule in comparison with the total number of cases of astigmatism was greater than we had hitherto believed. We reached this conclusion independently.

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[1] Lancaster, W. B.: *Tr. Am. Acad. Ophth. and Oto-Laryngol.*, 1915, p. 167.

LANCASTER'S CHARTS.

Lancaster's charts may be described as follows: Chart 1 is a square 65 by 65*cm* of white, smooth, unglazed paper, or so-called wedding stock, on which a circle 30*cm* in radius is drawn. At intervals of 10 degrees, radii are drawn from the periphery to within 10*cm* of the center. Strips of black velvet baby ribbon, known in the shops as No. 1½, 7*mm* wide, 20*cm* long, are glued to the cardboard, extending from the periphery to within 10*cm* of the center. The finished chart may then be compared to a rimless wheel with thirty-six spokes, having a clear circular center or hub 20*cm* in diameter. Chart 2 consists of a square made from the same stock, measuring 45*cm* on each side. A circle 36*cm* in diameter is drawn on this square and the periphery lightly marked at intervals of 10 degrees. A disk 36*cm* in diameter is cut out of the same stock and two diameters of black velvet baby ribbon are glued to the disk at right angles. The disk is then pivoted to the marked circle by means of a double-pronged staple, such as is used for binding legal papers. The disk may thus be turned to any desired position.

It will be seen that in many respects Chart 1 resembles the familiar sun-ray figure. But there are some points of difference which, in our opinion, are of extreme importance, rendering the charts the most valuable of any we have used for the detection of low grades of astigmatism.

In the first place, the charts are larger than those generally in use. The radii are longer, thus enabling the patient to select the most distinct line with greater ease. Second, the numbers corresponding to degrees of arc are drawn very lightly at the periphery and only for the purpose of aiding the examiner. In some other types of chart these numbers, which are not concerned in the test proper, are drawn so conspicuously as to distract the patient's attention from the radiating lines. In the third place, there is the exceedingly sharp contrast between the pure smooth white surface of the wedding stock and the soft yet intense black (literally, "velvet black") of the baby ribbon. One has only to compare this chart with the ordinary engraved or printed astigmatic chart to appreciate how superior it is in respect to contrast. It is surprising,

too, to observe that even after prolonged use, this chart does not become appreciably soiled—the smooth surface sheds dust very readily and the baby ribbon does not lose its velvety blackness. We have seen astigmatic charts in which the grime of the city had obscured all effective contrast and in which the black of the lines had faded to a rusty black or gray. In the measurement of higher degrees of astigmatism, it has been found of some advantage to have similar charts, but with No. 1 baby ribbon 5.5*mm* wide.

USE OF THE CHARTS.

We deem it of the utmost importance to secure a very accurate measurement of the dynamic refraction. We would not disparage for a moment the importance, nay, the vital necessity, of examining the refraction under complete cycloplegia in nearly all prepresbyopic patients, and, indeed, in many of those over the presbyopic age. Only in this way can information of importance to the patient and the oculist be secured, both as a present help in determining the immediate correction and as a guide to the subsequent management of the case. We are inclined to believe that many who employ cycloplegics habitually depend almost wholly on a knowledge of the static refraction and ignore the information, often very valuable, which is revealed by an exact estimate of the dynamic refraction. The reasons for this are twofold: First, the measurement of the dynamic refraction, even under the most favorable conditions, is a tedious process, requiring patience and care on the part of the examiner and the patient; the urge of a waiting-room full of impatient patients is not conducive to exactness in testing the refraction without a cycloplegic; second, the means at our command--keratometer, test letters, and the usual run of astigmatic charts, have not always revealed the existence of a low astigmatism.

The vast majority of astigmatic eyes exhibit this error in low degrees, and it is precisely in the detection and measurement of low degrees of astigmatism that Lancaster's charts possess their greatest usefulness.

We should like to dwell for a moment on the importance of preparing the patient physically and psychically for the

test that he is about to undergo. It is vastly better to deal with a patient in the early morning, preferably before he or she has encountered any of the stresses of the day. It is equally important that the mind should be calm, and the attention not distracted. During the test we debar other patients from the refraction room. If the visual acuity indicates that the error is H + Ah or Ah, plus spherical lenses strong enough to blur the letters to $\frac{6}{3 \cdot 3}$ or $\frac{6}{3 \cdot 5}$ are placed in a trial frame before the eyes. The screen bearing the test letters, which are illuminated by frosted mazda lamps, is at the end of a darkened corridor. As soon as the appropriate spheres are placed before the eyes, the lights are turned off and the patient told to look vacantly (that is, not to rivet the attention on any object) into the "dark distance." The examiner busies himself with other matters, while the patient continues to stare vacantly into the dark. There is no incentive for the accommodation to assert itself in the interest of clear vision, and we are convinced that more complete relaxation is thus secured.

After a few minutes the lights are turned on and the patient is asked to read, with both eyes open. It is not unusual to find that vision has risen to $\frac{6}{2 \cdot 6}$ or $\frac{6}{1 \cdot 6}$. The left eye is then covered with a blind and the vision of the right determined. The patient's attention is next directed to the wheel chart, and he is given plenty of time to observe whether any differences are observable in the appearance of the spokes. Certain spokes may be declared to be "heavier," a term that seems to mean one thing to one patient and another thing to another. For one, the meaning is "broader but less distinct"; for another the meaning is "black and clean cut." If the fogging is too great and the astigmatism low, the radii may all look equally blurred, so that it becomes necessary to reduce the sphere. This should be done by intervals of 0.12 D., and with each reduction the visual acuity should be taken and attention redirected to the wheel chart. In the presence of any astigmatism one line or, at most, an adjoining group of three or four lines will emerge clear and sharp. It is absolutely essential that the change in the strength of the sphere should be so gradual as not to throw any part of the focal interval of Sturm behind the retina. We emphasize this well-known fact,

because, as has been pointed out by Lancaster, one of the reasons for the failure to detect astigmatism with astigmatic charts is that the interfocal circle, which does not blur one line more than another, is, by reason of insufficient fogging, permitted to fall on the retina. Under these circumstances the lines, though blurred, appear uniform, and the examiner is apt to fall into the error of assuming that no astigmatism exists.

When one, two, or three adjoining lines appear more distinctly demarcated against the white background, we can feel assured that some astigmatism exists. If two adjoining lines are equally distinct it is almost certain that the correct axis lies between them. Thus, with the lines placed at intervals of 10 degrees, the chart will frequently give us the precise axis, that is, within 5 degrees, which, for most eyes, is within the limit of possible accuracy. The cross on the disk is then placed with one arm corresponding to the selected line, and gradual reductions with minus cylinders, at right angles to the selected line, are made until the other arm is rendered equally distinct. It may then become necessary slightly to reduce the spherical element in the interest of the sharpest acuity. In case of this reduction a retest with minus cylinders is made and the weakest minus cylinder which equalizes the arms of the cross is the measure of the astigmatism.

It must be admitted that this method is time consuming. The patient's attention is apt to flag, and it is wise to allow him some intervals of rest. But these are minor drawbacks; we feel well repaid for our time if we succeed in unearthing a low astigmatism which has eluded other methods of examination, including skiascopy.

Experience indicates that a fairly large number of cases of low astigmatism give no evidence of their presence to the shadow test. In low grades of astigmatism, it is often exceedingly difficult to be certain of the measurement to 0.12 of a diopter. We have therefore eliminated all cases which showed no more than 0.12 D. of astigmatism, at whatever axis.

In the determination of the exact axis, we have been largely guided by the patient's selection of the line on the astigmatic chart, but have often permitted him to rotate the cylinder to that position which rendered the smaller letters the sharpest.

While this expedient is of great value in astigmatism of 0.75 D. and upward, it is very difficult, in lower degrees, for the patient to be sure within 15 degrees of the precise position of the axis.

Our experience leads us to believe that inverse astigmatism is extraordinarily frequent in cases of chronic glaucoma. Such cases, as well as any examples of deformation of the cornea from injury or disease, pressure (chalazion), traction (pterygium), or operative corneal section have been rigidly excluded. Every patient included in our series has been examined ophthalmoscopically and the eyegrounds have been found normal.

In these cases we have not considered the question of muscle balance for the reason that, although the balance and ductions were determined in nearly every case, we were able to convince ourselves that the presence of low degrees of inverse astigmatism was without influence on the function of the extrinsic muscles. We believe many, if not the majority of the cases of low inverse astigmatism are lenticular in origin. This view is supported by the fact that in no less than seventy-one eyes with astigmatism against the rule the ophthalmometric measurement showed a total absence of corneal astigmatism.

There is a greater interference with visual acuity in the case of inverse astigmatism than in the case of an astigmatism of equal amount with the rule. One has only to render oneself artificially astigmatic with the rule and then artificially astigmatic against the rule (using the same cylinder) to appreciate the difference in distinctness of the same line of letters.

It by no means follows that every eye presenting inverse astigmatism should have this anomaly corrected. Elderly patients, who have worn spheres for many years and are well satisfied with them, do not take kindly to a cylindric correction. On the other hand, we are convinced that the addition of a low cylinder is often the means of abolishing symptoms which have persisted after the wearing of simple spheres. It is no unusual thing for patients to declare that while their former glasses, which were spherical, were helpful, they were still "conscious of their eyes." The introduction of the appropriate cylinder has been attended with the happiest results, often leading to the spontaneous expression, "These glasses are the best I have ever worn."

We must ever be on our guard lest the patient unintentionally misleads us. Our patients are of all grades of intelligence, hence misunderstandings are apt to arise. The success or failure of the test depends in no small measure on a correct understanding by the examiner of the patient's statements, which are often erroneous. Many do not seem to realize promptly the meaning of "horizontal" and "vertical," and in other ways fail to express clearly what they actually observe.

De Schweinitz[1] presents the results of a study of 100 cases of astigmatism contrary to the rule. All were examined under cycloplegia except when glaucomatous symptoms were present. He found nothing particularly characteristic of these inverse astigmatisms, so far as symptomatology was concerned, but reached the conclusion that when horizontal astigmatism exists, we are warranted in correcting fully every particle of the defect that we can render manifest.

Theobald[2] states that inverse astigmatism of even slight degree gives rise to pronounced asthenopic symptoms, and that its correction is attended with decided relief. Furthermore, an eye with this type of error is capable of "hiding it away," so that its detection is more difficult than the detection of perpendicular astigmatism. He believes that the relationship of inverse astigmatism and chronic glaucoma is more than coincidental.

Several writers, among whom are Steiger, Pfalz, and Schön, have noted a progressive diminution of perpendicular astigmatism and an increase in horizontal astigmatism as age advances.

The percentage of inverse astigmatism in school-children was found by Nordenson[3] to be only 1.3. Similar conclusions were reached by Hess on the basis of skiascopic examinations.

In regard to sex as a factor, Pflueger found inverse astigmatism to predominate in females, the ratio being 11:9. An even greater predominance of the female sex is indicated in de

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[1] De Schweinitz, G. E.: "One Hundred Cases of Astigmatism Contrary to the Rule," *The Journal A. M. A.*, 1891, xvii., 359.

[2] Theobald: "Is Astigmatism a Factor in the Causation of Glaucoma?" *Tr. Am. Ophth. Soc.*, 1888, xxiv., 126.

[3] Nordenson: *Ann. d'ocul.*, 1883, lxxxix., 110.

Schweinitz's series, as he found the anomaly present in sixty-nine females and thirty-one males.

The present study is based on a review of all eyes with astigmatism occurring in our private practice from July 1, 1916, to March 1, 1918, a period of twenty months. As stated in the foregoing, all eyes showing an astigmatism of not more than 0.12 D. have been excluded. We have also omitted the few cases in which the principal meridians were at 45 and 135 degrees. There were 1024 eyes which exhibited an astigmatism of 0.25 D. or over. Of these 741, or 72.3% were with the rule and 283, or 27.7% against the rule. The number of patients with astigmatism against the rule was 182, of whom 69 were males and 113 females. This refractive anomaly was present in one eye in 81 patients; it was present in both eyes in 101 patients.

Table 1 displays the incidence by decades. If this eighty-year period be divided into two groups of forty years each, eighty-nine patients belong in the first period and ninety-three in the second.

TABLE 1.—INCIDENCE BY DECADES OF ASTIGMATISM AGAINST THE RULE

Years	Number
0 to 10	0
11 to 20	21
21 to 30	37
31 to 40	31
41 to 50	50
51 to 60	22
61 to 70	13
71 to 80	8

Of the 182 patients, 114 were examined without cycloplegia and sixty-eight with cycloplegia. In the examination of the sixty-eight patients, homatropine, 3%, was used in fifty-one; atropine, 1%, in nine; scopolamine, 0.1%, in six, and euphthalmine, 2%, in two. We recognize, of course, that euphthalmine is not a cycloplegic, but have inserted the two cases here for convenience. Presbyopic correction was required in eighty-seven patients, a fact which explains the relatively large number of patients whose eyes were tested without cycloplegia.

In thirty-one eyes tested with drops, the shadow test

failed to reveal the presence of any astigmatism. These were all low astigmatisms associated with a high axial ametropia. It has been our experience that under these conditions low astigmatism often escapes detection by skiascopy. This may account for the failure of some oculists to recognize and correct certain low astigmatisms. Failing to find any skiascopic evidence of the presence of astigmatism, they do not pursue the inquiry any further, but content themselves with a spherical correction. In fifty-nine cases, patients were wearing or presented glasses prescribed by men whom we knew to be ophthalmologists. A neutralization of these lenses showed that in twenty-eight the inverse astigmatism had been recognized; in thirty-one it had not. Thirty patients showed us glasses prescribed by refracting opticians. Neutralization determined the fact that in only four cases was there any recognition of the existence of astigmatism against the rule. An ophthalmometric reading was taken in nearly every case. It is interesting to note that seventy-one eyes gave a reading of 0 and many others indicated low (0.25 or 0.5 D.) astigmatism with the rule.

TABLE 2.—TYPES OF REFRACTIVE ERROR OBSERVED IN TWO HUNDRED AND EIGHTY-THREE EYES

Refraction	No. of Eyes
H + Ah.	180
Ah.	40
M + Am.	35
Am.	7
Mixed astigmatism.	21

The symptomatology was variable and indefinite. The most frequent symptoms were headache, frequently occipital in type, indefinite ocular and retroöcular discomfort or pain, asthenopia and conjunctival and palpebral irritation. In the majority of cases there was an associated axial ametropia, so that it was impossible to be certain whether the symptoms may not have been due in part to hyperopia or myopia, as the case might be. In many patients previously given simple spheres, the addition of the appropriate cylinder was immediately recognized as a great improvement. The relief of symptoms, when the inverse astigmatism was the principal error, was often very striking.

CONCLUSIONS.

1. The percentage of inverse astigmatism is larger than is generally surmised.

2. The preponderance of females in the ratio of about 2:1 was a surprise to us, but is confirmed by the findings of de Schweinitz and others.

3. As has been noted by other observers, inverse astigmatism was not observed in children under 10 years of age.

4. Inverse astigmatism appears to be nearly as frequent from birth to middle age, as from middle age to old age.

5. Many patients tolerate a moderate inverse astigmatism up to the presbyopic age, when symptoms referable to the refractive error first manifest themselves.

6. The detection of inverse astigmatism is at times difficult with ordinary measures and care, often eluding the examining oculist and only in rare instances being detected by the refracting optician.

7. No special symptomatology can be attached to inverse astigmatism, the complaints being diverse and often indefinite.

8. Lancaster's charts have been of inestimable value in the detection of low grades of inverse astigmatism.

A CASE OF ACUTE RETROBULBAR NEURITIS IN A CHILD WITH INTESTINAL STASIS (CONGENITAL DILATATION OF THE COLON).

By Dr. MARK J. SCHOENBERG.

In the afternoon of December 5, 1917, a boy (Kenneth L.), 4 years old, was brought to the Presbyterian Hospital (Dr. Mason's service). He had high fever and had become entirely blind while eating his lunch that same day. His mother related that the child got up in the morning as bright as ever, had played with toys and appeared cheerful and normal in every respect. Just before lunch the mother showed him the picture of a horse and he made the remark that he could not see the legs of the animal. A few minutes later, at the table, he complained that he could not see his dinner. His look became stary but he did not appear sick. He was fed by his mother and ate with appetite. He had no headache, did not vomit, and complained of no dizziness. The face and extremities did not become pale; there were no twitchings or convulsions. He walked (when led) without staggering; his speech and hearing were normal as usual. The mother noticed that the child had high fever during the lunch. A day before he had had a bowel movement consisting of "soft brown stools." Urination was normal.

For the past four weeks the child had been coughing slightly and was given Stokes's expectorant. The medicine was refilled four days before and taken regularly until the day when blindness occurred, when he took the last three remaining doses (a teaspoonful each). The medicine had no ill effect, when taken two weeks before. Except for this mixture the child had taken no medicine, and there are no other drugs or poisons in the home. No drops had been used in the patient's eyes. He had had no falls or injury of any kind.

Family history. Parents and one other child well. Mother never miscarried and there was no history of chronic diseases in the family.

Personal history. Patient is the second child, full term, normal labor, strong and healthy. Bottle fed to thirteen months. No digestive troubles except occasional constipation. Always bright and active.

Previous illness. Measles in July, 1916. Whooping cough March, 1917. No history of tonsillitis, colds, rheumatism. No convulsions or fainting spells. Never had trouble with sight. Climbs stairs easily.

Examination on admission. General appearance. Well-developed, well-nourished child. Moderate dyspnœa and slight cough. Appears blind in both eyes. Expression dull. Occasionally twitching in fingers. Skin normal. Nose, throat, ears, normal. Thorax, lungs normal. Heart enlarged downward. A loud systolic murmur is heard at the apex, which is transmitted to the axilla. Abdomen moderately distended, tympanitic. Firm masses are felt in the left iliac fossa. No tenderness nor rigidity of the abdominal walls. Genitals normal. Extremities show occasional twitchings in the hands. Superficial lymph glands normal. Reflexes normal. No Babinsky, no Kernig. Temperature 102° F. The eyes were examined in the evening, about seven hours after the child had begun to complain of loss of vision. He was bright and could easily be induced to coöperate. He moved his eyes in every direction when told to do so, and showed no impairment of motility; understood very well when he was spoken to, and gave intelligent answers. He could not see even a strong electric light and made no attempt at winking when an object was brought quickly towards his eyes. The corneal reflex was present and normal. The pupils were dilated, equal, regular, and did not react to light. The ophthalmoscopic examination revealed a papillœdema of about 2 D., equal on both sides; the retinal veins were engorged, but the arteries normal in size. No hemorrhages or exudates.

At midnight the patient was quiet, the temperature had reduced to 99.6. The pupils were dilated and rigid to light.

Dec. 6, 1917. 12.15 A.M. Lumbar puncture. 10*cc* fluid removed for examination. Cells 4. Noguchi and Wassermann negative.

1 A.M. Colon irrigation. 1.45 A.M. Patient had a movement, consisting of *several quarts* (!) of very ill-smelling feces. 2 A.M. Pupils less dilated, react readily to light. Patient begins to see, follows the light, counts fingers, and distinguishes a bottle from a cup.

9 A.M. Pupils respond promptly to light; patient recognizes his father at a distance of several feet; distinguishes various objects.

The examination of the urine, blood Wassermann, blood

and throat cultures revealed nothing abnormal. The urine did not contain indican.

Chemical blood examination. CO_2 33.25 mgr. Blood sugar .118 mgr. glucose per 100*cc* of blood. Blood urea 0.025.

Blood count.	White bl. corp.	17,600
	Polymorphonuclears	66%
	Small lymphocytes	31%
	Mononuclears	1%
	Transitionals	1%
	Eosinophiles	1%

X-ray skull. Nasal sinuses negative.

Heart. Enlarged, especially to the right. Total transverse diameter 4½ inches.

Eye examination on Dec. 6. Pupils well contracted during sleep. As soon as the child woke up the pupils dilated, but the right pupil remained slightly larger than the left. Fundus. Both disks œdematous but less than the day before. Veins less engorged. The right disk is slightly more raised than the left. The vision, which could only be roughly estimated, consisted in ability to call out the name of a quarter, knife, pencil, when these objects were held at a distance of about two feet. Field of vision good peripherally for movement of hand.

Dec. 7. Again a very unusually large mass of feces after a high enema, mostly constipated, foul smelling masses. Patient feels well. Is cheerful and says he sees better.

Dec. 8. Vision improved, can distinguish a nickel from a quarter at about seven feet distance. Pupils slightly dilated, right slightly larger than left. Fundus. Right disk clearing up; slight œdema nasally. Left disk blurry throughout; swollen to about 1 D. Motility of both eyes normal.

Dec. 16. Boy behaves as if he has a central scotoma. If shown colored pictures of boys or girls he cannot point at the eyes or mouths, although he can readily tell which is a boy or a girl. When told to pick up colored beads or pieces of paper from the floor he will pick up the largest ones, and if urged to pick up the smaller ones he tries to perform his part of the game by feeling around and using the tactile more than his visual sense. He cannot point to where a small 5*mm* piece of white paper is, when held just in front of one of his eyes, but will see it when held to either side.

The patient continued to have very copious (several pints at a time), foul-smelling defecations, in spite of the restricted diet he had in the hospital.

Dec. 22. Vision: recognizes pictures of girls and boys at about six–eight feet distance.

Jan. 8, 1918. Temperature suddenly rose to 103°. Patient's vision seems to be less acute, but could not be measured properly on account of his general condition. A high enema brought about again a very large stool (characterized by Dr. Blood, in charge of the children's ward, as three–four times the amount passed by an average child of this age) and a prompt reduction to normal of temperature and improvement of vision.

An X-ray examination of the intestinal tract showed a redundancy of the sigmoid flexure and of the descending colon. The sigmoid rises to the 3d lumbar vertebra.

A chemical and microscopic examination of the stools by Dr. Dwyer (whom I wish to thank for his kindness) made on Jan. 9, 1918, "revealed a highly acid, extremely toxic stool with evidence of a mild mucous colitis. The bacteria were almost wholly Gram positive."

Jan. 24, 1918. Called at the H. Knapp Memorial Eye Hospital. General condition good. Pupils O. D.>O. S.; react promptly to light and accommodation. Vision: picks up with ease 2 sq. *mm* pieces of red and white paper from the floor and recognizes a penny and a nickel at twenty-feet distance.

Feb. 16, 1918. Fundus. Right disk temporally atrophic. Left disk atrophic and blurry above; inflammatory process not entirely subsided.

March 19, 1918. Vision about $\frac{6}{10}$ in each eye. Fundus same as before.

Summary. This is a case of optic neuritis (retrobulbar type) manifesting itself clinically by a sudden onset of blindness which began to recede very soon after the expulsion of a very considerable amount of feces. The course of the visual disturbance made the impression upon all, who saw and followed up the patient, that the condition was due to an autointoxication of intestinal origin. How near this diagnosis is to the truth is a matter for the reader to decide. We have no means nowadays of proving the existence of an intestinal autointoxication. The presence of intestinal stasis and the coincidence of an optic neuritis does not prove a causal relation between the two, but the rapid reappearance of vision after the very large movement of the bowels is very suggestive of such a relation.

If we think of the other conditions which might have pro-

duced in this child the bilateral optic neuritis we have to consider first, bronchitis, possibly an influenza which preceded by a few weeks the onset of the eye trouble; second, the possibility of the presence of wood alcohol in the tincture of opium contained in the Stokes's mixture given to the child for the cough. An infectious disease, like influenza, may give very rarely a retrobulbar neuritis by way of the blood or by way of a nasal sinusitis. In these cases there is usually found a star figure in the macular region and the spinal fluid shows the presence of a mild lymphocytosis (Knapp, *Medical Ophthalmology*), which were not present in our patient. Neither does the vision improve so rapidly after a colon irrigation or spinal puncture in influenza optic neuritis as it did in our patient. The presence of wood alcohol in Stokes's mixture is not probable considering the fact that it was obtained from a well known and reputable New York dispensary. Besides, wood alcohol if taken in sufficient quantity to produce complete amaurosis will give a number of other symptoms, like gastro-intestinal irritation and coma or prostration, which were absent in this patient.

Was the speedy return of sight after the successful colon irrigation due to the removal of the toxic feces only a coincidence? Did the spinal puncture just prior to the enema have anything to do with the return of vision? I do not think so. A spinal puncture may act beneficially upon the central nervous system by relieving momentarily an intracranial pressure and in cases of uremic amaurosis or in delirium tremens the removal of (a good deal more than 10*cc*) spinal fluid may under rare circumstances improve vision or delirium. But in such cases we deal with intoxication of the brain substance itself and increased intracranial pressure. In our patient there was no sign or symptom of increased pressure or disturbed cerebration and the blindness was due to a lesion in the optic nerve, not in the visual centers.

Finally—how could an intestinal stasis like that present in our patient cause an optic neuritis? The mere presence of intestinal stasis and putrefaction is not sufficient to cause the passage of poisons into the blood. The mucosa lining the intestinal tract is the first and a very effective line of defense against autointoxication. Even if intestinal poisons succeed

in passing into the circulation they are arrested and made innocuous by the liver, which constitutes the second line of defense. Finally when this barrier is broken down, there seems to be a third line of defense consisting of substances thrown into the circulation by the glands of internal secretion which have a detoxicating function upon poisons of whatever origin entering the blood stream. As we see it, the question of intestinal intoxication and its damaging effect upon certain tissues is not only a matter of absorption of toxic substances from the intestinal tract, but is a subject intimately connected with a number of other very complex problems.

In our case the dilatation of the large intestine (probably congenital) and the stasis and putrefaction of fecal matter must have injured the mucosa and allowed the continuous absorption of intestinal poisons. These must have overwhelmed the other detoxicating organs by their high toxicity, amount, etc., in order to reach the optic nerves. Why the products of intestinal putrefaction in this case affect the optic nerves, in other cases the uvea (see Dr. Dwyer's report in ARCH. OPHTH., vol. xlii., No. 3), is a question which future researches may answer.

I wish to thank Dr. H. H. Mason for permission to publish the report of this case from his service at the Presbyterian Hospital.

CYSTOTOME MANŒUVRE: EXTRACTION OF MATURE AND IMMATURE CATARACTOUS LENSES WITHIN THE CAPSULE.

PRELIMINARY REPORT, FIFTEEN OPERATIVE CASES.

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THE most desirable method of extraction of a senile cataract mature or immature is the one in which the lens in its intact capsule may be delivered with the least danger of loss of vitreous and minimum amount of trauma to eye tissues.

The writer has found that the lens can be rotated on its anteroposterior axis by a cystotome manœuvre and the zonule of Zinn disrupted in its entire circumference, freeing the lens within its capsule from its attachments without risk of loss of vitreous or perceptible additional traumatic effects to the usual extraction.

The intracapsular extraction in vogue at this time is Henry Smith's method in which forcible expression is employed to rupture the ligament of the lens, and even in the hands of the most expert there is a loss of vitreous recorded in a considerable number of the cases operated upon in this way. The post-operative reaction is in excess of the usual extraction because of the additional trauma requisite to dislodge the lens.

There were twelve mature and three immature cataracts in the last fifteen consecutive extractions of senile cataract operations and of this number thirteen lenses were delivered within the capsule. These gratifying results with the follow-

ing technique in the writer's mind warrant this preliminary report.

After the usual incision and iridectomy upward for the extraction of a senile cataract, the conjunctiva is grasped with fixation forceps two and one half millimeters from the corneal limbus in the lower nasal segment to steady the globe; the tip of the cystotome is then entered at the outer portion of the incision and passed blade flatwise in a downward and inward direction in front of the capsule, then behind the iris, until it has reached the locality of the lens ligament in its lower inner segment; the instrument is then turned at an angle of 90° to engage its sharp point into the capsular tissue near the zonule of Zinn; when this is done a quick jerky movement is executed circularly inward and upward in the direction of the zonule of the lens. The release of the lens in its capsule may be seen if the field of operation is watched closely. When this is accomplished the cystotome is carefully withdrawn blade flatwise, the lens delivered by pressure upon the lower portion of the cornea, pillars of the coloboma dressed down, and bandage applied.

The cystotome used is the type in which a spear point is fixed at the extreme tip of a round slender flexible shaft and at right angles to it. The sides of the spear point are equal and each measures 1*mm*. The entire length of the instrument is 123*mm* and consists of a shaft 27*mm* and handle 96*mm*. The shaft may be bent to meet the demands of the case to be operated upon.

The ligament of the lens was probably not entirely disrupted in the two cases in which the cystotome manœuvre failed because by pressure upon the lower corneal area by a spoon and pressure above the corneal incision at the same time the lens did not present. In these cases I reëntered the cystotome and did the usual incisions of the anterior capsule, delivered the lens but the capsule remained. In one other case succeeding these the lens failed to present also after the cystotome manœuvre and instead of incising the capsule as in the two cases just cited I entered the point of the cystotome in the ligamentary region at the margin of the outer pillar of the coloboma and made the same quick manœuvre with the cystotome but in the opposite direction, that is toward the

inner pillar of the coloboma. The lens was freed and presented readily in the corneal wound by gentle pressure upon the lower portion of the cornea and was delivered in its capsule. In the future I will try this before I proceed to the incisions of the anterior capsule.

Three of the cataracts of this series were immature and were delivered within the intact capsule. Two were delivered without accident. In expressing the other one the capsule burst when the lens was in the corneal incision and almost delivered. Some of the liquid clear cortex remained in the anterior chamber and the chamber was gently and thoroughly flushed. Four days afterwards a few small opaque lenticular spots were visible at the margin of the dilated pupil below but none appeared in the line of direct vision. There was no observable vestige of the capsule remaining.

The subsequent healing and convalescence of eyes operated upon with this technique differed in no particular from operations in which the lenses were delivered following incisions made into the anterior capsule. A failure to accomplish the disruption of the lens ligament by this cystotome manœuvre would not be serious because incisions may still be made into the anterior capsule according to stated methods and the extraction completed in the usual way.

Iritis did not develop and there was no loss of vitreous in any of the eyes operated upon in this series. The visual results with correcting lenses were as follows: $\frac{20}{15}$ in two, $\frac{20}{20}$ in one, $\frac{20}{30}$ in three, and $\frac{20}{40}$ in seven of them.

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A SIMPLE ELECTRICAL DEVICE FOR ILLUMINATING THE FIELD OF OPERATION IN OPERATIONS ON THE EYE, NOSE, AND THROAT.

By JOSEPH A. ANDREWS, M.D., F.A.C.S., Santa Barbara, Cal.

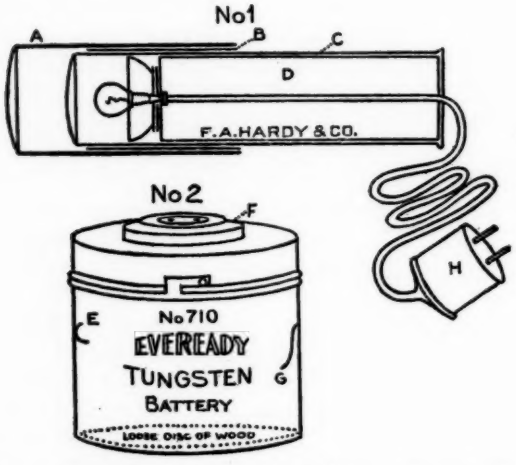
CONSULTING OPHTHALMIC AND AURAL SURGEON TO THE CITY HOSPITAL, NEW YORK.

(*With one illustration in the text.*)

THIS simple and inexpensive device is quite adequate for illuminating the field of operation in cataract extraction and other operations on the eye requiring concentrated light. Especially useful is one of these devices in operations for secondary cataract, and other operations on the eye where perfect illumination of the field of operation is so essential in enabling the surgeon to guide his knife and to carry out effectively the toilet of the eye, which is indispensable to satisfactory results.

No. 1 consists of an ordinary flashlight to which has been added the sliding brass tube A, carrying, at one end, a plano-convex lens by means of which the rays of light may be changed to suit the case. B is a strip of felt enveloping the barrel, which enables the tube A to slide smoothly over the parts beneath. For the one-cell battery supplied with these flashlights, I have substituted a core of wood through the center of which passes a wire which makes contact at one end with the lamp, and at its other end carries a cord of any desired length, connecting it, by means of a plug, with a 710 "Eveready" five-cell battery. The lamp used is a tungsten 6.2 V. which is obtainable of any electrical supply company. The receptacle for the battery I had made by a tinsmith. The lid of the receptacle has a bayonet fastening which insures

contact with the battery. The Bryant Jr. flush receptacle in the lid of the receptacle which receives the plug may be obtained of any electrical supply company. The instrument maker will supply a nickel-plated brass receptacle which will be more ornamental.



No. 1. A, Brass tube with plano convex lens: $3\frac{1}{2}$" long, $1\frac{11}{16}$" in diameter. B, Felt. C, Tube of flashlight: $8\frac{1}{8}$" long, $1\frac{5}{16}$" in diameter. D, Core of wood.

No. 2. Receptacle for battery: $3\frac{7}{8}$" deep, $4\frac{1}{16}$" in diameter. E, Brass to hold battery in place. F, Bryant Jr. flush receptacle for plug H.

The battery may be used in connection with Phillips's electric head light or any similar head light. The Phillips head light carries a lamp of a special make, not readily obtained, which costs $1.50. I have changed the socket in the head light to receive an ordinary 6.2 V. tungsten lamp[1] which costs 15c. to 25c. and which is entirely satisfactory for all operations on the eye, nose, and throat.

[1] F. A. Hardy & Co., 15 West 36th Street, New York City, will furnish these devices.

A VARIATION IN THE DISTRIBUTION OF THE NERVUS ABDUCENS IN MAN.

By THOMAS BYRD MAGATH, Ph.D.

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(With two illustrations in the text.)

THE eye muscles and their nerves present one of the most constant features of vertebrate anatomy. Neal (1914) writes:

"In the great majority of vertebrates the number and nerve relations of the eye muscles remain unchanged. Nature seems to have pursued with regard to them the policy of 'letting well-enough alone.' Their 'evolutionary potential' appears to be approximately zero."

From the dogfish to man one finds six pairs of muscles innervated by three pairs of nerves and the distribution, save in a few rare cases, is always as follows:

M. rectus superior
M. rectus medialis
M. rectus inferior
M. obliquus inferior } Nervus oculomotorius
M. obliquus superior—Nervus trochlearis
M. rectus lateralis—Nervus abducens

Some few deviations from the above arrangement have been pointed out in literature. Thus Allis (1897) states that in Petromyzon the abducens innervates the inferior as well as the lateral rectus, while the third nerve supplies only the rectus superior, rectus medialis, and obliquus inferior. Johnston (1905) says that in Petromyzon the "posterior rectus" (lateral rectus) is innervated by a separate slip of the motor trigeminus which corresponds to the so-called abducens. The other muscles are innervated in the usual manner. However, Neal

(1914) states that there is no abducens and no "posterior rectus" (lateral rectus) in this form and cites Johnston (1905), who does not make such a statement, as his authority. The final word has yet to be said, therefore, concerning the muscles and nerves in the Cyclostomes.

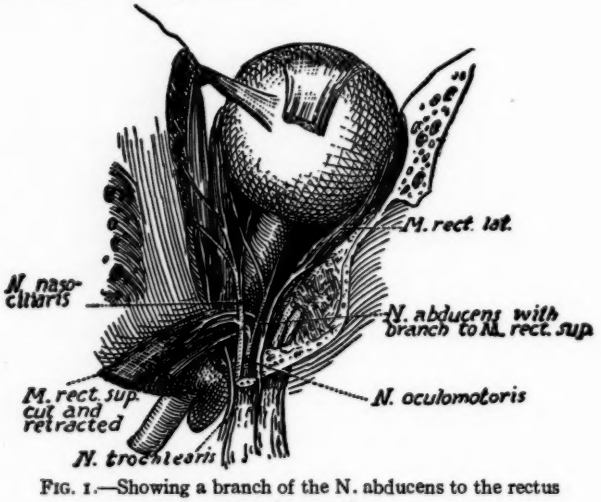

FIG. 1.—Showing a branch of the N. abducens to the rectus superior muscle.

In Astroscopus (Neal, 1918) some of the eye muscles are transformed into electroplaxes and in the reptiles and some mammals a retractor bulbi (oculi) is found as a derivative of the lateral rectus (Johnson, 1913, Fraser, 1915).

In man only a very few abnormalities have been reported in the innervation of the eye muscles. In Quain's *Anatomy* one finds the following statements concerning such variations:

"A communication between the third and sixth nerves as they pass the cavernous sinus has been described, but its existence has been denied by most recent observers. The upper division of the third nerve may have a communication with the nasociliary (Svitzer, Soemmering, Testut). The third nerve has been seen in a few cases giving a branch to the external rectus (Cruveilhier, Faesebeck, C. Krause), and in one instance a branch of the third supplied the place of the sixth nerve which was wanting (Generali). A filament to the

superior oblique muscle was seen by Arnold to pass through the lower part of the ciliary ganglion, and by Henle to pierce the inferior rectus.

"In one case the fourth nerve pierced the levator palpebræ superioris on its way to the superior oblique (G. D. Thane). The nerve has been observed in several cases sending a branch forwards to the orbicularis palpebrarum muscle, or to join the supratrochlear, the infratrochlear, or the nasal nerve. A communication with the frontal nerve is recorded by Berté, and A. F. Dixon found it in a human embryo.

"Absence of the sixth nerve upon one side is recorded, its place being supplied by a branch of the third nerve (Generali)."

In a recent dissection of the head of a male negro, a rather interesting abnormality was encountered on the right side. The abducens, at about the level of the upper division of the third nerve, sent a branch, which accompanied that of the oculomotor, to the superior rectus muscle. This branch of the abducens passed into the superior rectus after passing between the oculomotor and nasociliary nerves. The branch was as large as that to the same muscle from the third nerve. On the left side no variations were noted. The lateral rectus and the superior rectus were of normal size.

In trying to find an explanation for this curious phenomenon one may examine the embryology of the eye muscles and their early relations to their nerves. Neal (1918) sums up the data on the subject in the following words:

"From the first or pre-mandibular head cavity arise the muscles innervated by the oculomotor nerve, namely the recti superior, anterior (medial), and inferior, and the inferior oblique; from the second or mandibular myotome are differentiated the superior oblique muscle and the lateral portion of the external rectus; from the third head cavity develops the median portion of the external rectus muscle."

Figure 2a illustrates the normal origin and innervation of these muscles while Figure 2b shows the condition found in the specimen herein reported. One could not believe the muscles to have arisen on the right side of this specimen in any way save the normal. It is very hard to see how any other explanation for the variation can be had except along the line of the recent work of Parker (1910) who believes in

the primary independence of muscle and nerve. From the work of Harrison (1910) this hypothesis gains in weight, for

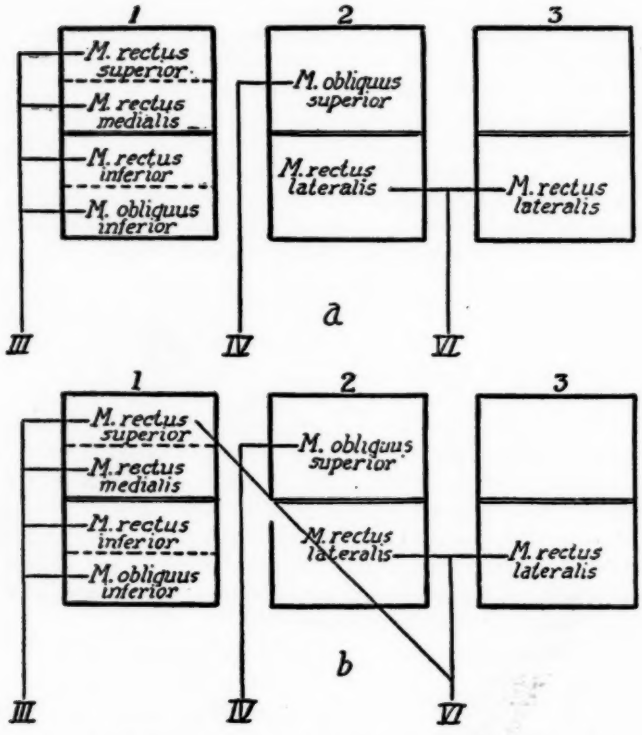

FIG. 2.—(*a*) Showing the normal method of innervation of the eye muscles. 1, 2, 3 are the first, second, and third myotomes, each of which is divided into a dorsal and ventral half. The muscles arising from each half are indicated.

(*b*) Showing the abnormal method of innervation in the specimen reported in this paper.

he has shown how isolated nerve filaments may grow into various types of tissue.

It is not unlikely, therefore, that this particular abnormality arose long after the eye muscles were differentiated and came

about as the result of some kind of stimulation to the nerve, which, as a response, sent out fibers to the external rectus. In the light of Harrison's observations one may accept such an explanation rather than assuming some embryological variation, and the conclusions of Neal on the origin of the eye muscles are in no way attacked by finding a case in which a nerve normally supplying a ventral moiety came to supply a dorsal moiety from another somite.

From the standpoint of the ophthalmologist such abnormal innervations are important, for they explain abnormal eye motions in certain cases and may be useful or detrimental in transplantations of the eye muscles, according to the variation present and the type of transplantation done.

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NOTE:—Since preparing the manuscript for this paper the author has received a note of explanation from Dr. H. V. Neal, in regard to the statement made concerning the eye muscles and eye muscle nerves of Petromyzon. Dr. Neal writes: "The statement in my '14 paper quoted in your letter of Sept. 19 which has just reached me here is very evidently a slip of the pen. What I should have written upon the authority of Johnston was that Cyclostomes have no Abducens nerve. This statement seems to me to be justified by all the facts which we now have." The author wishes to leave the matter at this point and refers the reader to the original papers for further discussion.

THE EYESIGHT OF THE NEGROES OF AFRICA.

By Dr. J. N. ROY.

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DURING a recent tour of study throughout the Dark Continent, where I visited twenty-two different colonies, I had the opportunity of examining the anatomical, physiological, and pathological condition of about five thousand negroes of one hundred different tribes, and it is the result of my researches on their eyesight that I intend to report in this paper.

After dealing with the question of refraction, vision during the day and night and accommodation, I will rapidly compare the eyes of the blacks with those of the other races of the earth.

In my work I used the de Wecker chart—built on the same principal as the Snellen type—in which the smallest characters are normally perceived at the distance of five meters. My subjects being illiterate, I had to use the special chart made up of a certain number of squares of different dimensions, where one side left out represents the opening of each square. In this way my examinations were very simple and precise. Even the least intelligent could easily indicate with his hand the opening of the squares.

I used skiascopy for the refraction. Under the circumstances, the difficulty of transporting the ophthalmometer would not have been repaid by the services obtained from it.

The African light is very bright and the atmosphere very clear. Experiments led me to the conclusion that vision in the

same person is better in Africa than in Europe. An emmetropic eye which, in the northern countries, has with good light a vision of $\frac{6}{6}$ or even $\frac{6}{5}$, can see in Africa one meter farther away, *i.e.*, a vision of $\frac{7}{7}$. This unity of $\frac{7}{7}$ will, in this paper, be considered as the normal vision of a European in Africa or in the tropical countries.

A glance at the geography and ethnology of the Dark Continent shows that the different races came in contact according to certain circumstances worth mentioning. The negroes of the Senegal Soudan intermarried with their neighbors of the north, the Berbers and the Arabs and their offspring the Moors peopled chiefly Mauretania. Those of the northeast were gradually absorbed by the Egyptians, who were always at war with the Ethiopians, and their union with the Fellahs gave origin to the race of the Peuhls and the Foulahs who live in the neighborhood of the Senegal River, the upper part of the Niger and in the Fouta Djalon in French Guinea. The Arabs of Yemen made repeated incursions into Africa, crossed the coast of Somalis, and reached Harar the capital of the country of Gallas in Abyssinia where they remained in possession for a long time.

The negroes who came in contact with these different races inherited from them many diseases, chiefly syphilis and trachoma. I shall, at a later date, take the opportunity of dealing with this question of syphilis on that continent. Smallpox also made terrible ravages chiefly where the means of communications are naturally easier than in the wild forest. The same may be said of purulent ophthalmia. In a general way the eyes of the people living north of the fifth degree latitude north are not as good as those living south, on account of the lesions due to purulent ophthalmia, variola, trachoma, and the constant irritation of the sand.

Strabismus, due usually to corneal scars or amblyopia from other sources, is also more frequent in the northern part.

I made a complete tour around the African continent penetrating at many points even to a great depth. My conclusions are in a word that the people of the south were first infected by those of the north, who in turn had been contaminated by the Indo-Europeans.

It would be outside the sphere of this paper, even on the

point of view of the union of races and the resulting contagion, to deal longer with this continent which in the past probably bound Asia and the south of Africa and of which the islands of the Sonde are the vestiges. The Hottentots and the Bushmen with their Mongolian blood are a living proof of this hypothesis. The last descendants of these tribes, almost extinct nowadays, live grouped together chiefly on the frontiers of Rhodesia, German Southwest Africa, and the Portuguese Angola.

Upon close examination of the eyes of the negroes, one is astonished at the considerable amount of pigment contained therein. At times it is even found on the ocular conjunctiva opposite the external canthus. The iris and the choroid are saturated with it and even in certain cases there is a physiological condition of melanosis of the soft palate and of the gums.

Refraction shows that their eyes are almost all emmetropic, provided there is no alteration of the cornea due to previous diseases. My statistics have given me the following results. Simple myopia is found in the proportion of about 1.5 per 100 and then it is always of a mild degree. I never had to use a glass higher than four diopters and this was with albinos. In passing it is worthy of note that this pigmentary anomaly is far more frequent in Africa than elsewhere. In Nigeria I saw a family of the tribe of Yorubas composed of five children, one dark and four albinos. It would have been interesting to find out, on the point of view of progeny, if the albinos intermarried, but in spite of a most careful inquiry, it was impossible to establish that fact, on account of the way they are treated in their own villages. At places they have no prestige, at others they are considered as unlucky, sometimes sacrificed at their birth or regarded as fetish.

Myopic astigmatism is found in about the same proportion as simple myopia, *i.e.*, 1.5 per 100 and I never had to use a cylinder higher than -2.5 to correct the error.

There is an absence of keratoconus and keratoglobus in Africa.

Simple hypermetropia and hyperopic astigmatism are found in about 2.5 per 100. The highest degree of hypermetropia was corrected by a sphere of $+ 3.5$ and astigmatism by a $+ 2.5$

cylinder. I saw but few cases of compound astigmatism and those chiefly among albinos. I never had a case of mixed or irregular astigmatism.

All these percentages are based on cases with absolutely transparent corneas. It goes without saying that alterations of the cornea due to diseases are met with in Africa as elsewhere. These percentages are based on an average of the whole continent. Where schools of instruction have long been established, they may be too low, but at places where almost every one was emmetropic, they are certainly too high. Practically all cases of ametropia were corrected by very weak glasses.

In a general way, I have been surprised at the excellent eyesight of the negroes. In rare cases, I have even observed that a correction of two to three diopters of either hypermetropia or myopia could improve their vision up to $\frac{9}{5}$ and even $\frac{11}{5}$. The highest normal vision recorded was one of $\frac{20}{5}$ and this was in a man affected with sleeping sickness. I shall, in a later paper, deal with this disease and show the great resistance of the negroes' eyes to the human trypanosomiasis. My statistics show that the average vision of the natives of Africa is $\frac{12}{5}$. More than any other race, have they the faculty of seeing to a great depth in water. In spite of the most careful researches, even in eyes suffering from diseases, I have never observed any alteration of the color vision or hemianopsia.

It is with pleasure that I mention my gratitude to our confreres of all nationalities with whom I came in contact. Their organization of all kinds have been a great help to me in my researches.

The negroes have a great power of vision in the dark. I made a series of experiments to compare their sight at night with ours. The subjects, both Europeans and natives, were first refracted and all ametropic or diseased cases were eliminated. To be understood by all, I had to proceed in the following simple manner. I made with white paper certain figures representing a square, a cross, or letters like L, I, V, T, etc. These figures were placed on a blackboard. The group to be examined stood far from these objects, the Europeans on the right and the natives on the left. A period of one

minute was allotted at each distance. If at the end of that time none of the group could see, all moved forward one step and so on. When any one could recognize the object, he was led aside and had to describe in some way the figure seen. If he was right, the distance at which he could see was measured; if not, he had to rejoin the group. In this way, I calculated the distance at which everyone could recognize the object. I repeated the test with the different figures. My experiments, repeated frequently and at different places, show that the negroes see at night from two to four times better than the whites. Admitting this fact, what is the explanation? A number of enucleations has given me the material for histological researches chiefly on the retina. The comparative anatomy of the eye shows that it is in the rods of the retina that lies the difference between the diurnal and the nocturnal animals.[1] Schultze had expressed the opinion that the latter had no retinal cones. Later this was found to be erroneous, as most of the reptiles, who are nocturnal, have more cones than rods. Up to now the only difference found is that the nocturnal animals have longer and thinner rods than the diurnals. I paid special attention to the examination of the retina. Unhappily the histologists I consulted found nothing new on the subject. There was a larger amount of pigment in the eyes of the negroes than in those of the whites, but rods in both races have the same form. We are forced to believe that their excellent vision in the dark is due to their greater use of their eyes at night compared to us. I have noticed the same thing with the Indo-Chinese, the Malays, redskins, whose vision at night is very good, and even among the Hindoos whose origin is Aryan.

I paid particular attention to their power of accommodation. It was easy to have positive facts on account of the presence, at different places, of scholars who could read fairly well. My researches on the subject show that the natives of the Dark Continent have a better power of accommodation than the whites, according to the standard given by the figures of Donders which I will discuss later on. A study of a series

[1] J. N. Roy. "Anatomie et physiologie comparées de l'œil et de ses annexes," *Archives d'Ophtalmologie de Paris.* Juillet–Août, 1912.

of fifty observations from my statistics show without doubt the existence of this superiority and, besides, corroborates all our previous statements. These observations are practically alike and were all made on emmetropic cases. To get the *punctum proximum* I used the smallest characters of the de Wecker chart. The optometer with thread would have given results hard to control.

Obs.														
"	I.	Man	tribe	Ouolofs	16	years	amp.	acc.	0.07	cent.	= 14.28	diopt.	Em. V	= 14/5
"	II.	"	"	"	18	"	"	"	0.082	"	= 12.19	"	Em. V	= 12/5
"	III.	"	"	Bambara	17	"	"	"	0.08	"	= 12.5	"	Em. V	= 13/5
"	IV.	"	"	Mandingue	29	"	"	"	0.107	"	= 9.34	"	Em. V	= 12/5
"	V.	"	"	"	15	"	"	"	0.068	"	= 14.7	"	Em. V	= 10/5
"	VI.	"	"	Bambara	20	"	"	"	0.085	"	= 11.76	"	Em. V	= 15/5
"	VII.	"	"	Malinke	22	"	"	"	0.088	"	= 11.36	"	Em. V	= 12/5
"	VIII.	"	"	Soussous	24	"	"	"	0.09	"	= 11.11	"	Em. V	= 12/5
"	IX.	"	"	"	30	"	"	"	0.103	"	= 9.7	"	Em. V	= 11/5
"	X.	"	"	"	23	"	"	"	0.09	"	= 11.11	"	Em. V	= 12/5
"	XI.	Woman	"	Fantis	14	"	"	"	0.067	"	= 14.92	"	Em. V	= 16/5
"	XII.	"	"	Apollonienne	14	"	"	"	0.07	"	= 14.28	"	Em. V	= 12/5
"	XIII.	"	"	Fantis	13	"	"	"	0.065	"	= 15.38	"	Em. V	= 15/5
"	XIV.	"	"	Accra	15	"	"	"	0.072	"	= 13.88	"	Em. V	= 11/5
"	XV.	"	"	Achantis	22	"	"	"	0.084	"	= 11.9	"	Em. V	= 9/5
"	XVI.	"	"	"	18	"	"	"	0.77	"	= 12.98	"	Em. V	= 14/5
"	XVII.	"	"	Yoruba	29	"	"	"	0.106	"	= 9.43	"	Em. V	= 11/5
"	XVIII.	"	"	Ibos	20	"	"	"	0.088	"	= 11.36	"	Em. V	= 14/5
"	XIX.	"	"	Egbas	22	"	"	"	0.08	"	= 12.5	"	Em. V	= 15/5
"	XX.	"	"	Yoruba	25	"	"	"	0.102	"	= 9.8	"	Em. V	= 10/5
"	XXI.	Man	"	Minas	23	"	"	"	0.085	"	= 11.76	"	Em. V	= 14/5
"	XXII.	"	"	"	23	"	"	"	0.08	"	= 12.5	"	Em. V	= 17/5
"	XXIII.	"	"	Evhe	25	"	"	"	0.087	"	= 11.49	"	Em. V	= 18/5
"	XXIV.	"	"	Dagomba	19	"	"	"	0.07	"	= 14.28	"	Em. V	= 11/5
"	XXV.	"	"	Fons	19	"	"	"	0.075	"	= 13.33	"	Em. V	= 10/5
"	XXVI.	"	"	Bassa	23	"	"	"	0.073	"	= 13.69	"	Em. V	= 14/5
"	XXVII.	"	"	Malumba	21	"	"	"	0.07	"	= 14.28	"	Em. V	= 15/5
"	XXVIII.	"	"	Kumba	24	"	"	"	0.082	"	= 12.19	"	Em. V	= 13/5
"	XXIX.	"	"	Malumba	21	"	"	"	0.074	"	= 13.51	"	Em. V	= 10/5
"	XXX.	"	"	Bassa	18	"	"	"	0.07	"	= 14.28	"	Em. V	= 12/5
"	XXXI.	"	"	Loango	16	"	"	"	0.07	"	= 14.28	"	Em. V	= 10/5
"	XXXII.	"	"	Yacoma	13	"	"	"	0.067	"	= 14.92	"	Em. V	= 13/5
"	XXXIII.	"	"	Batetela	16	"	"	"	0.065	"	= 15.38	"	Em. V	= 10/5
"	XXXIV.	"	"	Azande	15	"	"	"	0.075	"	= 13.33	"	Em. V	= 12/5
"	XXXV.	"	"	Bangala	16	"	"	"	0.075	"	= 13.33	"	Em. V	= 11/5
"	XXXVI.	"	"	Yacoma	15	"	"	"	0.07	"	= 14.28	"	Em. V	= 15/5
"	XXXVII.	"	"	Basonge	14	"	"	"	0.065	"	= 15.38	"	Em. V	= 11/5
"	XXXVIII.	"	"	Bangala	15	"	"	"	0.068	"	= 14.7	"	Em. V	= 12/5
"	XXXIX.	"	"	Bakumu	15	"	"	"	0.075	"	= 13.33	"	Em. V	= 10/5
"	XL.	"	"	Basoko	14	"	"	"	0.062	"	= 16.12	"	Em. V	= 12/5
"	XLI.	Woman	"	Baluba	14	"	"	"	0.068	"	= 14.7	"	Em. V	= 12/5
"	XLII.	"	"	Mongo	18	"	"	"	0.074	"	= 13.51	"	Em. V	= 11/5
"	XLIII.	"	"	Yacoma	17	"	"	"	0.07	"	= 14.28	"	Em. V	= 15/5
"	XLIV.	"	"	Basoko	15	"	"	"	0.066	"	= 15.15	"	Em. V	= 17/5
"	XLV.	"	"	"	20	"	"	"	0.08	"	= 12.5	"	Em. V	= 10/5
"	XLVI.	"	"	Bangala	14	"	"	"	0.06	"	= 16.16	"	Em. V	= 13/5
"	XLVII.	"	"	Bapoto	16	"	"	"	0.065	"	= 15.38	"	Em. V	= 12/5
"	XLVIII.	"	"	Mongo	16	"	"	"	0.068	"	= 14.7	"	Em. V	= 14/5
"	XLIX.	"	"	Basoko	19	"	"	"	0.07	"	= 14.28	"	Em. V	= 11/5
"	L.	"	"	Loango	15	"	"	"	0.063	"	= 15.87	"	Em. V	= 10/5

If we compare these figures with those of Donders's chart, we see to what point the amplitude of accommodation in the negroes is superior to that of the whites, although I must say that the difference is not as marked as it looks. I regret very much I had to limit my researches to the period of life between

13 and 30 years. Younger than that, my subjects could not read sufficiently to give definite answers, and after that I could not trust their age. The negroes cannot give exact answers about their birthday. At times they will mention a certain number of years before or after a definite event in the history of their country, and from this event, as well as we know it, we have to deduce their age. I regret it so much more as it would have been interesting to know when presbyopia develops among them. However our limited statistics with subjects whose age was well known, show that it develops a little later than in the whites. It is but logical on account of their excellent power of accommodation and the rarity of latent hypermetropia. This fact is worth mentioning because according to some authors among whom are H. Campbell Highet of Singapore, and J. Santos Fernandez[1] of Cuba, who have made some experiments in their own country, presbyopia would appear from five to six years sooner in the tropics than elsewhere, and this not only among the Europeans who have been residents for a certain time, but also among the natives. According to them the high temperature, causing a general depression of the organism, and indirectly a diminution of the power of accommodation, would explain this phenomena; besides the way of living and the feeding of these different people could also have something to do with this condition. It is not so with the negroes. Moreover I must say that the population of Singapore is composed of many nations in which the yellow element represented by the Chinese, the Japanese, and the Malays predominates. In Cuba there are Spaniards, Creoles, Chinese, Americans, and the other residents have lost, either as a result of their prolonged stay or through intermarriage, a part of the characteristics of their own race. These anæmic whites or yellow peoples often affected by malaria, or born from diseased parents, are from every point of view far less resistant than the blacks whose organism by heredity is used to the life in the tropics.

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[1] J. Santos Fernandez, "Influencia del clima de la Isla de Cuba en las enfermedades de los ojos," *Cronica Medico-Quirurgica de la Habana*, September, 1893.

"La presbiopia en la Isla de Cuba," *Cronica Medico-Quirurgica de la Habana*, Mayo, 1898.

In Africa I was surprised at the excellent power of accommodation compared to the figures of Donders and upon my return to Canada, I thought it would be interesting to make a control test of these figures. From schools of both sexes and from my private and hospital practice, I examined a great number of persons of different origin whose ages varied from 10 to 60 years. My results lead me to say that the amplitude of accommodation among the whites is greater than the Donders chart would show. In these experiments the de Wecker chart was used and the patients were asked to read, with one eye at a time, the smallest characters at the shortest distance. My conclusions are as follows: from 10 to 15 and from 50 to 60 years, my figures correspond to those of Donders; from 15 to 20, there is an increase in the amplitude of accommodation of one diopter; from 20 to 30, of two diopters; from 30 to 40, of three diopters; from 40 to 45, of two diopters, and from 45 to 50, of one diopter. Emmetropic cases only were examined, as the myopics and the hypermetropics would have given results either higher or lower than the Donders chart which represents only the physiological accommodation.

If we examine, in a general way, the vision among the different races of the globe, we see that it varies greatly in individuals of the same origin. To avoid repetitions and especially to remain on purely scientific ground, I shall divide, as I did in a paper on Ozæna, delivered before the French Congress of Oto-Rhino-Laryngology, in May, 1914,[1] the different peoples of the earth, from an ethnological standpoint, into three large families: the white, the black, and the yellow. Indeed, if we consider carefully the characteristics of the Malays and the redskins, we find that the representatives of those two races have had the Mongolians for ancestors. The color of their skin, the shape of their eyelids, the development of their malar bones, the flattening of the base of their nose, the thickness of their hair, and generally speaking their facial expression, are all arguments in favor of this theory. Moreover, we find in the language of certain Indian tribes of South America, a number of expressions very similar to some Japanese words, and in Mexico, ruins of temples have been found with

[1] J. N. Roy, "Ozæna among the Various Races of the Earth," *The Laryngoscope*, St. Louis, September, 1917.

symbols which are undoubtedly vestiges of those who adorned the shrines of Buddha.

It is not my intention to speak at length of the vision of the whites, it is too well known for that; I will simply point out the most interesting facts not yet mentioned and the results of my observations when their blood is mixed with that of negroes or of Mongols.

It is admitted that in Europe the errors of refraction depend on the races and on the degree of instruction, and as an example of that, we see that myopia is more frequent in Germany than elsewhere, while the Latins and chiefly the Anglo-Saxons in spite of their refined and advanced culture, are less predisposed to it. The same is noticed among the yellow peoples not only of Asia but where ever else my many trips have permitted me to study it locally.

Myopia quite frequent in Japan and China is met with almost exclusively among the educated class and in spite of what certain authors say, it is not a national defect. Hypermetropia is met with a little more frequently.

The Malays, Filipinos, Hovas, Esquimaux, Laplanders, and the redskins are like the negroes, almost all emmetropics, and Steiner[1] has the same opinion of the population of Java.

My statistics on the ametropia of the mulattoes and the half-breeds show that the vision in both has about the same characters. Seventy-five of the refracted cases among the mulattoes are astigmatics. In detail the percentage is as follows: 50% of hypermetropic astigmatism both simple and compound, 25% of myopic astigmatism simple and compound, 20% of hypermetropia and 5% of myopia. Dr. Jones[2] of Newport News, Virginia, in a paper on refraction of the half-breeds comes to the same conclusion. His figures show 78% of astigmatics of which 51% is of the simple or compound hypermetropic type, and 27% of the simple or compound myopic one. There is 19% of hypermetropia and 3% of myopia. The word "Indians" in this paper means half-breeds

[1] L. Steiner, "Coup d'œil sur 3104 cas de maladies des yeux chez les Malais," *Geneeskundig Tydschrift voor ned. Indie*, xxxvi., I.

[2] C. P. Jones, "A Study of One Hundred Refraction Cases in Indians Fresh from the Plains," *The Journal of the American Medical Association*, July 25, 1908.

only. It does not include the redskins, for nowadays these are seldom met with in their pure condition. To study them carefully, one has to go, as I did, to Mexico or to Central or South America.

No irregular or mixed astigmatism was found among the mulattoes or the half-breeds and all the corrections were of a slight degree.

Some authors have put forth the opinion that the depth of the orbit might have some influence on the development of the eye in its anteroposterior diameter and consequently affect the refraction. To deep orbits would correspond myopia and to shallow ones, hypermetropia. Moreover they think that the orbital diameter is in relation with the shape of the head and that the more dolichocephalic is the cranium, the deeper is the orbit and vice versa. We know that in a general way, the yellow peoples are more or less brachycephalic, the whites mesaticephalic, and the negroes almost all dolichocephalic. My prolonged studies among the main races of the earth has shown me the falsity of this theory. Outside of the anomalies of refraction, due to abnormal curvature of the lens or to previously existing diseases which, by softening the posterior segment of the eye, alter its anteroposterior diameter, there is no rule to explain the causes of ametropia other than direct heredity. These hereditarily weak eyes succumb to the effect of fatigue brought on by delicate work, corneal lesions, defective light, and the constant use of poor printing. Moreover according to Broca's measurements of the orbit the Dutch with their shallow cavities are frequently myopics, while the Esquimaux with their large orbits are almost all emmetropics.

The vision of the different peoples is also proportionate to their intellectual development. Taking the white race as a point of comparison, we see that the eyesight of the Chinese and the Japanese is similar to ours and of all the different branches of the yellow race, it is the worst. The Esquimaux and the Laplanders, living in the northern countries, are next in scale with a vision superior to that of their ancestors. The Filipinos, Hovas, Malays, and the redskins have still better sight.

Warm climate seems to have a certain influence on the

development of the ocular pigment. In individuals of the same race born in different places, more pigment is met with in the tropics than in the cold countries. As a result of this, there is a larger absorption of light rays and an improvement of the sight, although I must say that the intensity of the light plays a great part.

Studying the moral and the physical condition of the mulattoes and the half-breeds, we find that they inherited from the whites all their defects and even their patrological atavism. When the good qualities exist—which is exceptional—they are always poorly developed. Their eyes are not as good as those of the redskins and the negroes, but may be compared to ours.

The power of accommodation of the yellow race is—as in the whites—superior to the figures of Donders. My statistics show it about equal in both races. The same may be said of the mulattoes and the half-breeds.

In concluding, let me once more call to your attention, the marvelous visual apparatus of the negroes, as compared with that of the other peoples who live in a more or less primitive state, or who have reached the highest degree of civilization. After careful researches, I came to the conclusion that the higher the human being rises in the social scale, the more his brain develops at the expense of the organs of sense, chiefly of vision and of hearing, both of which can be estimated mathematically.

For the whites, their mode of life has a deleterious influence on their physique. The hygiene of the sight and hearing is defective on account of the use of strong artificial light and injurious noises. Diathesis is transmitted from generation to generation. In general, feeding is too heavy and too rich. Excesses of all kinds ruin the organism, and the needs of instruction require prolonged and tiresome studies.

None of these are found among the natives of Africa. They live the simplest life required by the most urgent needs of nature and they have no ambition. Of all human beings the blacks are without doubt the ones representing the lowest step of civilization. Their eyes and ears are developed to the highest possible degree of physiological function. Next in the scale are the yellow peoples of the islands of Oceania, then

those of Indo-China, the redskins of Mexico, of Central and South America, the Laplanders, the Esquimaux, and lastly the Chinese and the Japanese. The whites are the last in the scale with a poor vision compared to the other races, and even among them it is influenced, as I have said previously, by the degree of instruction and heredity.

The ocular pigment seems to have a definite influence in the perception of the objects and I believe that in the negroes, it is one of the main agents of their excellent vision. It absorbs some rays of light and neutralizes the ones that are irregularly reflected on the retina.

With the civilization brought to them by the Europeans who now control almost the whole of Africa; with their mode of living changed chiefly in the increased absorption of a more or less adulterated food and of the alcoholic beverages—which nowadays they even make themselves;—with the advance of instruction in schools and workshops; with at places the use of artificial light, it is a question how long will these natives keep up their visual power, at present so perfect.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By JOHN M. WHEELER, M.D., Secretary.

MONDAY EVENING, APRIL 15, 1918. MARTIN COHEN, M.D., CHAIRMAN.

Dr. W. E. Lambert presented two cases that he had operated on for **congenital dislocation of the lenses**—brother and sister. The mother also had dislocation of the lenses, but not sufficient to interfere with her vision. The boy before operation had $\frac{1}{100}$ vision in the right eye and $\frac{20}{200}$ in the left. The girl $\frac{20}{70}$ in the right, and fingers in the left. Four discissions were made on each eye in both cases, and the entire treatment and subsequent absorption of the lenses lasted about six months. The result in each case was most satisfactory, as the lenses were entirely absorbed. The girl's vision is $\frac{20}{30}$ plus in each eye, with + 12.00 spherical. The boy has $\frac{20}{30}$ V. O. D. and $\frac{20}{30}$ V. O. S., with the proper correction. An interesting feature about these cases is that they are both able to read ordinary print with the glasses which are used for distance. Dr. Lambert also presented another case of congenital dislocation of the lenses, on which he had done two needlings in the right eye. The lens was in the process of disintegration. There had not been the slightest complication in any of these cases.

Dr. Geo. H. Bell showed a patient 48 years of age who had been operated upon for **traumatic pulsating exophthalmos.** This patient was presented before the section at the meeting held February 18th. Dr. F. W. Murray ligated the right common carotid at the New York Eye and Ear Infirmary on February 26th and the patient was in bed for one month after

operation. The bruit has disappeared, the exophthalmos is decidedly less, and Dr. Bell hopes for still further improvement.

Dr. BELL showed a case of **iridocyclitis** which he thought was due to **dental infection.** Patient, age 48, was first seen on March 16, 1918. There was marked ciliary tenderness and injection, and the anterior chamber was nearly filled with blood. Through this blood could be seen a contracted pupil and an exudate in the pupil. Tension was slightly minus. The eye looked so suspicious of a foreign body that an X-ray was ordered, which proved to be negative. Wassermann was negative. On examining his mouth, Dr. Bell found that he had two loose teeth and severe pyorrhea. One tooth was extracted at once in the clinic, and the patient was referred to a dental clinic. His vision when first seen was hand movements at three or four inches. The patient was ordered into the New York Eye and Ear Infirmary where he got hot bathing and atropin treatment in addition to having his dental foci cleared up. One month later, tension normal, pupil dilated, blood and exudate all absorbed, and patient now has vision of $\frac{10}{20}$. This patient had no internal treatment.

Dr. BRUDER said that he had not seen a case of iritis which he considered due to trouble with the teeth. This patient has a history of inflammation of his joints of eight months' duration. He thinks there may be some cause other than infection from the teeth in Dr. Bell's case.

Dr. CRIGLER said that he had treated a patient who had suffered from iritis and secondary glaucoma throughout one year. He had been treated by different methods, including iridectomy, without relief until his teeth were all extracted. There was immediate improvement in the eye condition. The inflammation subsided and there has been no relapse.

Dr. LAMBERT had the impression that tooth infection was now well recognized as a cause of iritis.

Dr. TENNER thought that there might be a latent gonorrhea in this case. In his experience gonorrhea has occasioned more iritis than syphilis.

Dr. STEINBUGLER in a study of fifty-three cases found that a high percentage of iritis cases were due to tooth infection. Good results were obtained from vaccines.

Dr. BELL said in reference to this eye condition being due to rheumatism as one of the members present had suggested, he thought that all agreed that rheumatism was due to either the teeth or tonsils, and as this man had no tonsils, we would naturally think of a mouth infection in case of rheumatism. Sir W. Lang, who has gone into the etiology of two hundred cases of iritis in his practice, gives 6% of them due to syphilis; tuberculosis 11%; general affection 8.5%; other causes 25%; and pyorrhea 37% and gonorrhea 11%. In seventy-four of two hundred cases, the iritis was due to defective teeth and stumps. In this patient of his, the improvement was so great with clearing up of his mouth infection that Dr. Bell is forced to conclude that the iridocyclitis was due to oral sepsis. The patient's eye was in such a state of inflammation when first seen that it looked exactly like an eye with a foreign body in the globe, and treatment of his teeth was only a routine matter, which is done in Dr. Bell's clinic in all interesting or obscure cases.

Dr. MARK J. SCHOENBERG presented a case of **paralysis of associated movements** in a patient with syphilis. The patient, 40 years old, seamstress, American born, was infected with lues at the age of 18 years. She began to suffer from accommodative asthenopia and diplopia 8 years after the infection. At the age of 34 she was compelled to wear bifocal lenses, although myopic. When she first came to the Herman Knapp Memorial Eye Hospital the diagnosis was made of paralysis of associated movements upwards and to the left, insufficiency of convergence, and paralysis of accommodation. The neurological and serological examinations revealed several signs of cerebrospinal lues. The localization of the seat of the lesion for these various groups of oculomotor disturbances and the vascular distribution in the oculomotor centers were taken up in detail in the presentation of the case, and the conclusion was reached that the seat of the lesion was in the right anterior corpus quadrigeminum and in the nucleus medialis. Since the condition shows no tendency to progress and no active changes are present in the spinal fluid there is no indication for treatment.

Dr. KAUFMAN SCHLIVEK presented a case of the **retinitis proliferans** type which presented a remarkable ophthalmo-

scopic appearance. Antonio A., 25 years old, previous history negative except that at the time and for one year before he discovered that the right eye was blind he was working in a mine (possibility of trauma). Seven years ago the patient had a foreign body on the cornea of left eye and when he closed it noticed that he could not see with right eye. Vision in the right eye is fingers at one foot, up and temporally. Cornea, iris, anterior chamber, and tension are normal. The lower nasal quadrant of the nerve head can be seen with the ophthalmoscope and its margin is not sharp. The rest of the nerve head is covered by a mass which is irregularly cone-shaped and extends directly anteriorly to about the center of the globe. This mass is white and fluffy and on it are numerous vessels; some are dark red with no perivasculitis (new formed vessels), others with very marked perivasculitis. Some of these vessels extend backward on this mass to its base and then turn on the retina; some extend from the apex a short distance posteriorly and then swing over to the retina. Where these latter vessels continue on the retina it is decidedly elevated and from these vessels extend directly backward numerous very fine arborescent vessels. At the lower nasal quadrant of the nerve head the vessels are normal. A little above and to the nasal side of the nerve head there is a wide grayish membrane with some new formed vessels on it. This mass is connected with the large one. From the apex of the cone-shaped mass is another smaller grayish nonvascular one which is narrow for a short distance and then suddenly ends anteriorly in a knob. This knob has three extensions: a short narrow one below, one nasally, and one above. The latter two spread out into diffuse fibrillated areas and apparently extend over to the ora serrata. There is perivasculitis along several of the retinal vessels. Peripherally especially above and temporally there is chorioretinitis. In the macular region there are pigment changes. The left eye is normal. Wassermann and Noguchi tests are negative. Urine is normal. Blood pressure 90*mm* Hg. X-ray is negative. Transillumination is negative. No tuberculosis. This case corresponds to the descriptions of retinitis proliferans but nowhere is there any mention of this peculiar arrangement of the vessels.

Dr. JOHN M. WHEELER presented an **unusual case of**

persistent hyaloid artery. The patient, a young man 24 years of age, first learned of poor sight in his right eye a few months ago when he was drafted and rejected by an examiner on account of his defective vision. The right eye is highly myopic and yields vision of $\frac{8}{200}$. In the vitreous there is a strand which is attached to the optic nerve head and which has the characteristic appearance of a complete persistent hyaloid artery, without blood. The interesting peculiarity about this anomaly is that although the strand has the characteristic opacity at its anterior end which is usually seen at the posterior pole of the lens, and although the strand is a fixed body, there is no attachment to the lens. The anterior end is probably about *2mm* behind the lens and there is no apparent opacity on the capsule. Very fine strands can be seen radiating from the anterior end of the persistent hyaloid artery but they cannot be traced far from this body as they are exceedingly delicate. Dr. Wheeler's theory to interpret this condition is that originally this strand existed as an ordinary complete persistent hyaloid artery attached to the posterior capsule of the lens, and that in the development of the high myopia and with the constant lengthening of the globe the strand became detached from the lens but retained fixation in the vitreous by fine threads which may possibly be attached to the zonule and the ciliary body. The left eye is normal.

Dr. WILLIAM MCLEAN (by invitation) read an interesting paper on "Experimental Studies in Tonometry" (published in this number).

Prof. E. A. GREEN, Professor of Physics at the Boys' High School of Brooklyn, said that he had examined Dr. McLean's testing apparatus and was satisfied that it works in perfect accord with the simplest principles of hydrostatics. He said there was found an average variation of *15mm* between the Schiotz tonometer readings and those of the mercurial manometer.

Dr. G. DEWAYNE HALLETT said that because of his conviction that Dr. McLean had devised a scientific instrument which accurately measures in millimeters of mercury the intraocular tension he was glad that there developed in his service three cases appropriate for enucleation upon which these experiments could be made. In all these demonstra-

tions upon the human eye in situ, after Dr. McLean had inserted the canula into the globe, to the steady maintenance of which he gave his entire attention, it was Dr. Hallett's function to apply the footplate of the tonometer accurately to the cornea. An assistant read off the position of the marker on the scale. Another maintained the manometer in a proper position under Dr. Holzapfel's direction, and his guidance was taken from the location of the air bubble in the tube. Another assistant noted the pulsations of the manometer fluid as being synchronous with the heart's action. All this division was for the purpose of avoiding error. The readings were remarkably uniform, and wonderfully confirmed by experiments upon animal eyes, in situ and also enucleated. The excellent features of the instrument are: a longer reading arc, a direct reading scale, the proximity of the scale and the footplate, and the avoidance of weight shifting.

Dr. SCHOENBERG said that it would be interesting to know whether the lowering of intraocular tension which Dr. McLean reports under general anæsthesia is caused by a corresponding reduction of the general blood pressure. Dr. Schoenberg asked if Dr. McLean used cocaine, as this lowers intraocular tension.

Dr. MCLEAN replied that he had not made observations to determine the effect of cocaine on the tension in human eyes. He always uses holocaine as he has known of two cases of acute glaucoma from cocaine instillation. In one case cocaine was given for the removal of a chalazion and acute glaucoma followed.

Dr. LAMBERT asked if there was a constant difference between readings from the Schiotz and McLean tonometers.

Dr. MCLEAN replied that there was found to be a sliding scale of difference ranging from seven to twenty-five.

MONDAY EVENING, OCTOBER 21, 1918. MARTIN COHEN, M.D., CHAIRMAN.

Dr. M. URIBE-TRONCOSO presented a case of **tubercular endovasculitis of the retinal vessels with degeneration of optic nerve and retina.** The patient, 32 years old, a sailor, well built and healthy appearing, who at the end of July, 1918, noticed a blurring of vision in the left eye, rapidly coming to a total

blindness in four or five days. Being in France at that time, he was interned in the hospital for one month, his condition being diagnosed as "neuroretinitis" and a treatment of salvarsan and mercury instituted. The patient came back to this country in the middle of September and when first seen gave a history of chancroid two months before the beginning of the eye trouble. No other diseases were reported, his general health having always been good. On examination the left eye was found to be entirely blind. Externally there was only a slight pericorneal injection. The dilated iris was attached to the capsule of the lens by numerous synechiæ, mostly thread-like, and the rim of the iris adherent to a great extent, in the upper part of the pupil, by a whitish exudation. Media clear. Optic nerve hazy, covered by a white veil, with blurred margins and surrounded by a white narrow ring. Its level was slightly depressed, about one diopter.

A striking feature was the total absence of the arteries and veins upon the disk. On the borders some of the larger vessels could be detected; the lower temporal artery as a whitish cord and the upper temporal and lower nasal as strings of white glistening patches of a beaded appearance. In some places it was easy to detect between the patches, and also in the extreme periphery, the vessels transformed into white cords. Upon the disk, in the inner and upper part, small newly formed vessels radiate from the center like a fan. Around the papilla a complete ring of white glistening patches, linear or round, closely resembling the appearance and arrangement of a retinitis circinata, but encircling the papilla instead of the macula, could be detected. They were separated from the disk by a zone of a dark red color, in which small round hemorrhages were visible. The macular region, and in fact the whole eye ground, was covered with white round glistening spots about the size of a pin head and larger, generally arranged in groups. The choroid is normal, and no pigment is visible elsewhere.

Careful systemic examination detected a mitral lesion, but no other disease could be made out. Wassermann was negative twice in the blood, and also in the spinal fluid. Urine was normal. In view of these negative findings an injection of 1*mg* old tuberculin was made and after twelve hours a weak positive reaction was obtained. A new injection of 3*mg* and

another of 5*mg* gave very slight general reactions, but elicited a local reaction in the eye in the form of a hemorrhage in the upper and outer part of the disk. This hemorrhage invaded the vitreous and was surrounded by a hazy zone. A last injection of 10*mg* tuberculin produced a rise in temperature of one degree, and an enlargement of the hemorrhage into the vitreous, new vessels developing afterwards in the vicinity.

The interest of this case lies in the total occlusion of the retinal vessels and the degeneration of the nerve and retina. The author first thought of a gumma or tubercle of the optic nerve, but the vascular conditions looked so preëminent as to be considered the primary cause. The complete endovasculitis was probably induced by the sudden discharge into the blood of the central artery of a great number of tubercle bacilli, which produced septic emboli. This accounts for the rapid loss of sight. Then the nerve became inflamed and an exudate developed around the papilla. That there was not a sudden stopping of the circulation in the central artery is proved by the normal color of the fundus, the lack of the cherry red spot or its sequelæ, and the report of the French surgeon. Another interesting feature of this case is the formation of the ring of white spots around the disk, on the macula, and in the periphery of the eye ground. That they are related to the endovasculitis obliterans and not to the toxæmia seems to be a pretty certain conclusion, especially if we bring together the findings in this case with those of Dr. Cohen's case of Kroenlein operation for tumor of the optic nerve, presented at this meeting. He saw Dr. Cohen's case before the operation and only found in the left eye a secondary optic atrophy without any involvement of the retina and choroid. After the removal of the growth, the optic nerve and the short posterior ciliary arteries having been severed, the eye ground presented a very remarkable picture: in the vicinity of the semi-emptied retinal arteries, and closely following their course, a great number of white, glistening round and elongated spots could be seen, sometimes isolated, sometimes coalescing in large patches with festooned margins. These white spots had the same characteristic features of those observed in the case of endovasculitis, and also resembled closely those found in albuminuric retinitis and retinitis circinata. As it is certain

that in the case of Kroenlein operation there was not any general infection or toxæmia to account for the degeneration of the retina, the white spots starting right after the stopping of the circulation in the retinal vessels, it seems rather fair to ascribe them to the vascular disturbance with exclusion of any inflammatory condition whatsoever; the deficiency in the blood supply producing in the very sensitive retinal tissue a granular or fatty degeneration.

Dr. GEORGE H. BELL presented two cases of **nodular keratitis,** in a woman, age 50, and in her daughter, age 16. The woman complained of progressive deterioration of sight without pain or inflammation. The woman seemed to be in good health. The girl did not complain at all. The woman had V. O. D. $\frac{20}{60}$, V. O. S. $\frac{20}{70}$, with correction $\frac{20}{40}$. The girl V. O. D. $\frac{20}{30}$, V. O. S. $\frac{20}{30}$. This is a family degeneration of the cornea, and Fuchs considers the affection to be of the nature of a dystrophy, to be recognized by the deposition of substances insoluble in the tissue juices.

The rare corneal affection, which has characteristics similar to those of punctate keratitis, manifests itself in the formation of numerous small corneal opacities, the largest of which usually occupy the central portion. When examined with the loupe, the opacities are seen to be composed of numerous minute rounded or irregularly formed grayish spots located in the superficial layers of the cornea. (Some of the spots in my cases are deeply situated.) In the woman, rather large irregular patches appear at the pupillary area which result from a coalescence of small opacities. Between the opacities the cornea appears dull and hazy, and over the affected site the epithelial layer is somewhat elevated; the peripheral portion is generally not involved. Opacity occurs in both eyes simultaneously. It is a slow and chronic disease and starts early in life. Collins and Mayou state that hyaline material has been found in these patches. The little girl has the "*lattice-shaped variety.*" There is present, together with the spots, a network of minute lines in the cornea. Slight inflammatory symptoms make their appearance now and then, while the spots themselves very gradually increase. There is no anæsthesia of the cornea in either case. The Wassermann is negative in both cases.

Etiology is very obscure, but I think it is due to some form of toxæmia. Fuchs recently reported two cases he had, one in a man, age 50, and the other patient was a man, age 29. Fuchs operated upon both of these subjects. One was improved and one was not. Of course, in my cases, no operation is indicated or advised.

Dr. GUTTMANN said that Dr. Bell's cases were of interest on account of the fact that the disease attacked two members of the same family.

Dr. DAVIS referred to a case in a man 43 years of age. He said that the disease developed at the age of 25. There were numerous punctate spots situated in the center of the cornea in a perfect circular arrangement 4*mm* in diameter. The cornea was not anæsthetic. He said that the cause of such a condition is still unknown.

Dr. GEO. H. BELL presented a boy, age 14, with a piece of "gold" in the lens. Vision began to fail two weeks before he went to the New York Eye and Ear Infirmary. V. O. D., hand movements, V. O. S., $\frac{10}{20}$. No pain; no redness. At about 7 o'clock on cornea you can see scar where foreign body entered; on dilating pupil you can see foreign body in lens which has a gold luster. You can even see the yellow color in the daylight. He was working on a foot-press with gold metal and a chip flew off and struck him in the eye. This gold he was using for making rings. Patient did not experience any pain at the time of accident. There must have been some alloy in the gold as the patient now has a siderosis. He has never had any tension, and no inflammation. Dr. Bell expects to remove the piece of gold as well as the cataract next week. He was not able to get the patient's consent to the operation until the siderosis appeared, which was quite recently.

Dr. L. W. CRIGLER presented two cases of **trachoma with corneal involvement relieved by Heisrath-Kuhnt operation.** The first case was that of a woman 47 years of age who developed trachoma in the left eye ten years ago. After three years' duration, in spite of medicinal treatment, pannus developed in the upper quadrant of the cornea, and transformation from ulceration to cicatrization was steadily going on in the conjunctiva of the upper lid. This eye was operated upon seven years ago. Within a few weeks after the operation the

pannus disappeared and all inflammatory symptoms subsided. There has been no recurrence of the pannus since and the patient's vision has materially improved. About two years ago the patient noted trouble developing in her right eye. In view of the fact that it was inconvenient for her to visit the hospital, she has been treated at another institution, but noting the eye getting steadily worse she returned to the Manhattan Eye, Ear, and Throat Hospital saying that she would like the same treatment for the right eye as was given the left seven years previously. This eye was operated on a month ago and the blood-vessels in the cornea which could be seen extending almost to the pupillary border have almost entirely disappeared and with them the other inflammatory symptoms.

The second case was that of a young man who was operated on two years ago. His vision was reduced to light perception and the entire cornea was infiltrated in each eye. This young man states that he was carried before a class of students by one of our prominent ophthalmologists in another institution who stated that his vision would never improve. The patient now has $\frac{20}{200}$ vision in the right eye and $\frac{20}{100}$ in the left. The pannus has entirely disappeared but there is a permanent slight opacification. Every symptom of his trachoma has been relieved, and all treatment has been stopped. For six months after the operation he was kept under observation and treatment which consisted of mild astringents and dionin.

Dr. KNAPP said that the important feature of any operation is its late result. In this connection it may be interesting to note that Kuhnt's successor in Kōnigsberg does not do the "combined excision" operation.

Dr. DAVIS condemned the operation as being wrong in principle and said that he thought that such an operation should never be done. He said that he had seen a case recently in a young boy in which there was some interference in motility when the patient looked downward.

Dr. WOOTTON said that he could not see why there should be any aversion to the operation in view of the satisfactory results obtained and reported. He said that the main criticism which some expressed was due to faulty technique of the operator and should not be counted against it. Dr. Wootton said that

he and his associates at the Manhattan Eye, Ear, and Throat Hospital had operated on about two hundred of these cases during the past eight years and that the results have been so uniformly satisfactory as to convince the most skeptical.

Dr. COHEN said that so strongly was he convinced of the efficacy of the treatment that he did not wait for the formation of pannus or even the cicatricial stage of the disease before recommending the operation.

Dr. CRIGLER said that he was not quite so radical. He waits until there are evidences of beginning corneal involvement. He said that it was to be expected that if the operation is put off until the cartilage and conjunctiva have reached the degenerative stage, little good will come from any operation.

Dr. MARTIN COHEN presented a case of **striated keratitis.** A. H., age 35, married; has two children, both in excellent health. Wife has had three spontaneous miscarriages; one occurred after the birth of the first child, and two after the birth of the second child. The family history and previous diseases are of negative clinical value. General physical condition normal. Wassermann reaction positive.

The patient was first examined at the Manhattan Eye, Ear, and Throat Hospital three weeks ago. He then complained of disturbance of vision in the right eye, with photophobia, lacrymation, and slight pain. Vision was $\frac{1}{10}$ with no correction. Tension was normal. Pupil, circular, central, and reacted sluggishly to light. The aqueous was slightly turbid. Circumcorneal injection was marked. The upper and inner quadrant of the cornea was transparent; the lower and outer quadrant cloudy. This latter area, when examined with focal illumination and the stereoscope, consisted of nine grayish parallel striæ from 3-6*mm* in length and ½*mm* in width; the striæ radiating toward the center. The cornea is transparent between the striæ which space is 1*mm* in width. The striæ first appear about 1½*mm* from the corneal limbus. On a plane with the striæ two discrete linear exudates are readily seen.

Diagnosis: Striated keratitis, due to acute iridocyclitis. When the striæ are examined with transmitted light, using a 11 D. lens, one receives a diffuse reddish fundus reflex; the striæ are apparently fewer and are divisible into two thin grayish lines with a light reddish area between them. One also

recognizes in this area several horizontal striæ. The exudates are opaque.

In this case we are probably not dealing with a deep cellular infiltration, as is present in interstitial keratitis, but rather with a refractive condition of the cornea due to wrinklings in Descemet's membrane as described by Hess and Schirmer in similar cases and in experiments. These wrinklings are produced by a diffusion of abnormal fluid through the diseased endothelium into the deep parenchyma. This disturbs the tissue tension of the cornea and causes the development of the wrinkling. When the abnormal fluid is absorbed, the wrinkles disappear. The anatomical arrangement of the normal lymph spaces of the cornea and the appearance of the striæ as in this case preclude dilated clefts or lymph spaces being the cause of the striæ. In striated opacity of the cornea, as occurs after cataract operation, the striæ are located in the upper quadrant and run vertically from the corneal section; this condition is non-inflammatory, of a few days' duration, and is due to wrinkling in Descemet's membrane, as proved by various observers. Fuchs uses the term "striated opacity" in non-inflammatory conditions, while striated keratitis is applied to cases due to an inflammatory disease.

Treatment: Hot local applications, and the use of atropine three times a day. General treatment, anti-luetic. The condition is slowly improving.

Dr. COHEN presented a case of **tuberculosis of the conjunctiva**. Boy, age 8½, has never been seriously ill; family history negative. Physical examination—including X-ray examination of the lungs, differential blood count, and blood Wassermann reaction—normal. General tuberculin reaction was positive; however, numerous conjunctival smears were negative for tubercle bacilli or other specific organisms. Cultures were also negative for sporotrichon. Conjunctival scrapings inoculated into the anterior chamber of a rabbit, after two weeks, gave a negative result. The rhinological examination was negative.

The present eye condition, which is of ten weeks' duration, is solely localized to the left lower tarsal conjunctiva. The lid is decidedly thickened, and when everted (which is difficult) we perceive the conjunctival surface to be uneven, which is due to

numerous isolated raised papules about the size of the head of a pin; the spaces between the papules show necrotic tissue. Scattered over the same area are yellowish nodules simulating tuberculous nodules, varying in size from the head of a pin to a pin point. Some nodules are gradually invading the mucocutaneous border of the lid. The entire tarsal conjunctiva is frequently covered by a purulent exudate which when removed causes bleeding. The pre-auricular, cervical, and submaxillary glands are markedly enlarged solely on the side of the lesion. No other glandular enlargements.

The diagnosis was established by the clinical findings, the general tuberculin reaction, and the exclusion of conditions which simulate it. The disease is probably exogenous. The treatment consisted of frequent local irrigations with saturated boric acid solution, local applications of 1% silver nitrate, and general hygienic regulation with therapeutic tuberculin injections. Too short a time has elapsed to report any final results.

Dr. COHEN also presented a case of **tumor of the optic nerve (myxoglioma)** which is published in full in the ARCHIVES with photomicrographs.

Dr. ARNOLD KNAPP presented **two specimens of intraocular disease with anterior scleral ectasia.** Inflammatory conditions of the eyeball in which ectasias in the anterior part of the sclera develop are always serious, and as the diagnosis is not clear, the report of the two following cases in which this complication occurred may be of interest.

The first is that of a colored woman, J. T., aged 48, who came to the hospital in September, '17, stating that her eye had been inflamed for three months. The eye presented the symptoms of a severe iridochoroiditis with marked ciliary congestion, exudate in the pupillary space, and the formation of new vessels in the iris structure. Vision reduced to perception of light; no details could be observed in the fundus. The usual treatment brought about no particular improvement. The tests which were made were not positive. One month later she returned with a marked swelling of the sclera in the outer part. Tension was increased, inflammatory symptoms more pronounced, and the eye was painful. She consented to enucleation and on sectioning the eyeball the condition which the

specimen presents was found present. The microscopic examination revealed a tuberculous process.

The second case is that of a young man, aged 23, who was admitted to the army last fall with normal eyes. About three months later the sight in the left eye failed. Subsequent discharge with diagnosis of detachment of the retina from unknown cause. About a week before, May 23, '18, the eye became inflamed and painful. On examination there is a well-marked ciliary congestion and a pronounced scleral nodule at about the site of the internal rectus insertion. The anterior chamber is shallow, iris markings fair; oblique illumination shows a reddish mass in the nasal side of the eye ground covered with a small hemorrhage. Perception of light present. The Wassermann test proved negative, even after provocative salvarsan. The tuberculin test gave a marked arm reaction and fever, but no particular change in the eye. On June 8th the eye is enucleated and on opening the eyeball a well-marked sarcoma of the choroid is found present which has extended by perforating and thinning the sclera at the site corresponding to the internal rectus insertion. Slightly posterior there is another point in which the scleral tissues seem to have been traversed by the tumor process.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

BY MR. H. DICKINSON, LONDON.

An ordinary meeting of the Section was held on November 6th, the new President, Mr. W. T. HOLMES SPICER, F.R.C.S., occupying the chair.

PRESIDENT'S ADDRESS.

The PRESIDENT, after thanking the Section warmly for the honor accorded to him, proceeded to deliver an address, illustrated by many sketches, on **the causes of some of the appearances of the cornea after inflammation.**

After ophthalmia neonatorum, but little was sometimes to be seen except vascular channels, yet the stagnation of the lymph circulation might bring about slowly advancing changes long after the subsidence of all inflammatory appearances. Nettleship had described linear paths of clear corneal tissue in large diffuse lipomata, these latter having, probably, arisen after small spots in infancy. And Fuchs discussed the various linear formations existing in the cornea, suggesting that the lines were due to changes in the lymph system. In one of Sydney Stephenson's cases, the lines traversed a nebula with opacity, and there had been repeated attacks of corneal inflammation early in life. Story recorded lines of this character in interstitial keratitis, and he thought they were due to a clearing of the vessels. The picture presented varied according to the depth of the vessels concerned. It was uncommon for vessels to disappear and leave no trace: even though empty of

blood, they could be seen as silky threads on the surface, or as clear lines beneath the surface. The vessels in one layer of the cornea did not interfuse with those in another layer. The first necessity in the production of these conditions was a destruction of tissue. The contraction which followed that either interfered with nutrition, or interrupted the flow of lymph, leading to stagnation and the deposition of salts. Arcus senilis was more likely to occur in eyes which have had corneal ulceration.

Mr. Treacher Collins proposed and Mr. Lang seconded a cordial vote of thanks to the President for his address.

CASE.

Mr. HARLEY GOLDSMITH exhibited and demonstrated a case of **excessive dilatation of retinal veins and arteries** in one eye. When examined for admission into the Army a few weeks before, the examining surgeon said the vision of the affected eye was better than that of its fellow. The upper temporal vein was enormously congested, and the corresponding artery much dilated.

PAPER.

A paper was contributed to the Section by Dr. J. B. CHRISTOPHERSON and Major R. C. ARCHIBALD, of Khartoum, on **a case of primary nocardiasis of the lacrymal gland,** caused by a species of nocardia hitherto undescribed.

The authors said the lacrymal gland appeared to be one of the most remarkable glands of the body. Having but few disorders of its own, it fulfilled its destiny undisturbed by the disorders of the outside world, content to be left alone to administer its secretion to the great master-tissue the eye. The causal agent in the case here recorded was a germ or fungus which grew readily aerobically and produced arthrospores.

The patient, a male aged 22 years, was admitted to hospital suffering from a swelling of the right eye, and inability to open it. The duration of the case had been three and one half years. Neither scar nor wound was to be seen. The eyelids could not be everted, and when raised, a thick yellow discharge

poured out, and the whole conjunctival surface of the upper lid appeared to be rough and granular with deeper ulceration in parts. The swelling was painless and of a doughy consistency, though without œdema. There was a bulging forwards of the fornix of the conjunctiva. No lymphatic glands were enlarged, and the sight was unaffected. The eyeball itself was not invaded, nor were its movements involved. Neither photophobia nor lacrymation was present. Under chloroform, the growth was dissected away. It appeared to have commenced in the lacrymal gland, and to have spread into the upper lid, invading the tarsal cartilage. It extended also somewhat between the eyeball and the bony orbit along the ducts to the lacrymal gland.

Sections showed the morbid histology commonly associated with a fungal infection. A portion of a grain flattened between a slide and cover-glass showed that it was composed of the typical nocardial bacilliform hyphæ, with rounded bodies or spores, the whole being held together in a dense matrix. The paper described the reactions in various media, and gave an account of the mycology. So far as experimentation had gone, it did not seem to be pathogenic to the gray monkey.

Lieut.-Col. R. H. ELLIOT commented on the rarity of actinomycosis in Madras, especially in Madura where the disease generally was extremely common.

Mr. LESLIE PATON expressed the gratitude felt by the Section for the paper, for the specimens, and the precautions taken by the authors to ensure its safe arrival.